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SURGERY OF THE UPPER ABDOMEN

DEAVER AND ASHHURST

VOLUME I

SURGERY OF THE
STOMACH AND DUODENUM

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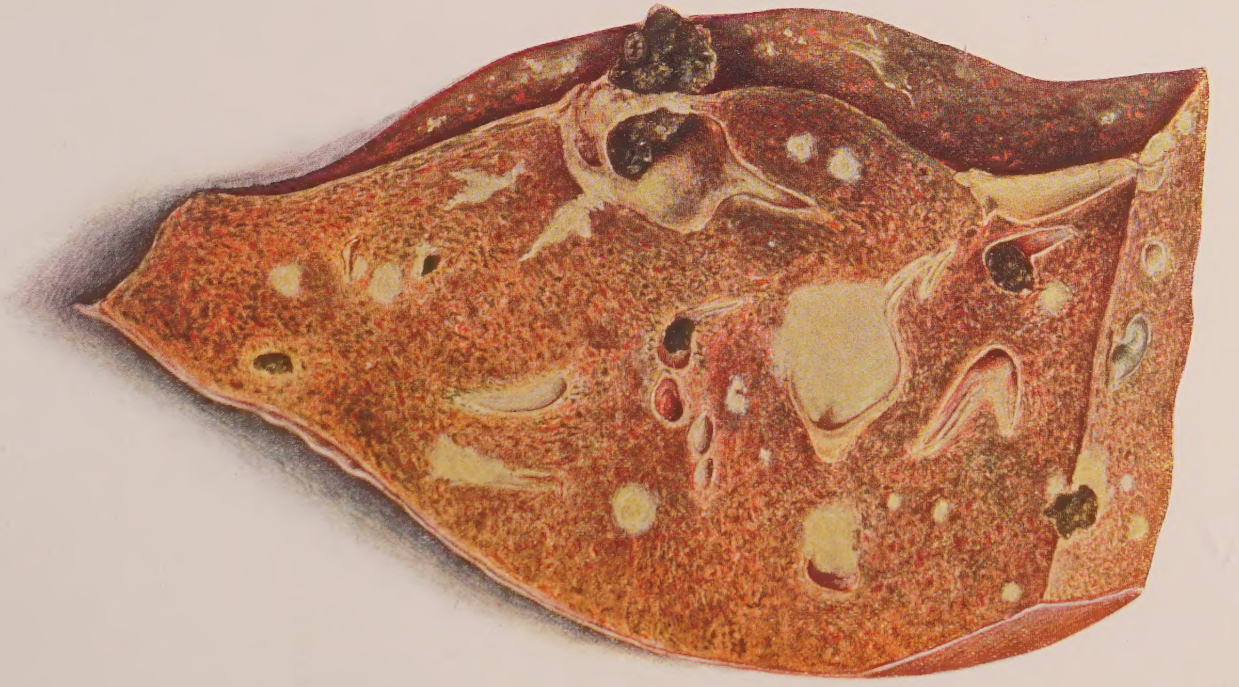

Suppurative Cholangeitis with Multiple Abscesses of the Liver. (Case History, page 174.)

SURGERY OF THE UPPER ABDOMEN

IN TWO VOLUMES

BY

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VOLUME II

SURGERY OF THE GALL-BLADDER, LIVER, PANCREAS AND SPLEEN

WITH 52 ILLUSTRATIONS

PHILADELPHIA

P. BLAKISTON'S SON & CO.

1012 WALNUT STREET

1914

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THE · MAPLE · PRESS · YORK · PA

PREFACE

In presenting to the profession the present volume, which completes their work on the Surgery of the Upper Abdomen, the authors desire to express their high appreciation of the very cordial and favorable manner in which the first volume has been received.

That an interval of more than four years has elapsed since the publication of the first volume is to be explained by the great mass of literature, not only recent, but also in a sense classical, which it is necessary for anyone to peruse who wishes to be thoroughly informed on the surgery of the gall-bladder, liver, pancreas and spleen. It has been the endeavor of the authors to supplement in this way their personal experience; and they believe they are thus enabled to furnish their professional brethren with what they hope may be regarded as authoritative sources of information and opinions which may be said to represent the crystallization of the views at present held in these important departments of surgery.

The authors have to thank Dr. A. D. Whiting for his very painstaking and accurate work in compiling the statistics derived from patients operated on at the German Hospital; for valuable criticism and assistance in the preparation of the text; and for the preparation of the index. Their thanks are due to Dr. J. R. Freeland, formerly Resident Physician at the German Hospital, for assistance in the preparation of the chapter on the Spleen; and to Miss Edna Patterson for tireless and enthusiastic work in preparing the manuscript for the press. The illustrations have all been drawn by Mr. Chas. F. Bauer.

J. B. D.
A. P. C. A.

September 24, 1913

TABLE OF CONTENTS

Chapter		Page
I.	Surgical Diseases of the Biliary Tract:	
	General Ætiological, Pathological and Diagnostic Considerations	1
	Gall-stone Formation	8
	Typhoid Infections of the Biliary Tract	17
	Jaundice as a Symptom	28
II.	Surgery of the Gall-bladder and Bile-ducts:	
	Deformities, Anomalies and Malpositions	39
	Cholangeitis and Cholecystitis	44
	Cholelithiasis	64
	Pathology	64
	Causes	84
	Symptoms and Diagnosis	93
	Treatment	110
	Obstruction of the Bile-ducts	122
	Strictures of the Bile-ducts	123
	Obstruction from Causes outside of the Ducts	139
	Biliary Fistula	144
	Intestinal Obstruction from Gall-stones	152
III.	Surgery of the Liver:	
	Anomalies of Size, Shape and Position	157
	Abscess of the Liver	164
	Cirrhosis of the Liver	182
	Hydatid Cysts	189
	Non-parasitic Cysts	203
IV.	Tumors of the Liver, Gall-bladder and Bile-ducts:	
	Benign Tumors of the Liver	206
	Gummata of the Liver	208
	Tuberculoma of the Liver	210
	Sarcoma of the Liver	210
	Carcinoma of the Liver	211
	Cystic Degeneration of the Gall-bladder	217
	Carcinoma of the Gall-bladder	218
	Carcinoma of the Bile-ducts	221
V.	Injuries of the Liver and Biliary Passages	235
	Rupture of the Liver	237
	Gunshot Wounds of the Liver	244
	Stabwounds of the Liver	247
	Injuries of the Gall-bladder and Bile-ducts	249
VI.	Surgery of the Pancreas:	
	Historical	254
	Congenital Anomalies, Displacements, etc	256
	Pathogenesis of Infections	259
	General Diagnostic Considerations	274
	Acute Pancreatitis	288

Chapter		Page
	Subacute Pancreatitis	307
	Chronic Pancreatitis	313
	Pancreatic Calculi	346
	Injuries of the Pancreas	351
VII.	Tumors of the Pancreas:	
	Carcinoma	353
	Sarcoma	367
	Cysts	368
VIII.	Surgery of the Spleen:	
	Anatomy	383
	Physiology	386
	General Consideration of Enlargements of the Spleen	390
	Movable Spleen	396
	Cysts of the Spleen	400
	Tumors of the Spleen	403
	Tuberculosis of the Spleen	404
	Abscess of the Spleen	404
	Splenic Anæmia	408
	Injuries of the Spleen	421
	Results of Splenectomy	427
IX.	Technique of Operations:	
	General Considerations	440
	Operations on the Gall-bladder and Bile-ducts	440
	Abdominal Incisions	441
	Cholecystendysis	447
	Cholecystostomy	448
	Cholecystectomy	454
	Choledochotomy	458
	Cholecystenterostomy	469
	Operations on the Liver:	
	Hepatopexy	471
	Transpleural Hepatotomy	472
	Hepatectomy	475
	Operations on the Pancreas:	
	Pancreatectomy	477
	Pancreato-enterostomy	480
	Operations of the Spleen:	
	Splenectomy	481
	Splenotomy	482
	Splenopexy	482
Index of Names		485
Index		491

LIST OF ILLUSTRATIONS

Figure		Page
	Suppurative Cholangêitis, with Multiple Abscesses of Liver	Frontispiece
1.	Mass of Gall-stones Conglomerated and Pressed into the Shape of the Gall-bladder	13
2.	Perforation of the Gall-bladder during Typhoid fever	21
3.	Hour-glass Gall-bladder	43
4.	Hydrops of the Gall-bladder	52
5.	Diagram to show Various Sites of Biliary Calculi	76
6.	Extreme Contraction of the Gall-bladder, the Result of Long-standing Cholecystitis	81
7.	Palpation of the Gall-bladder by "Thumb Pressure" under the ninth Costal Cartilage	102
8.	Bimanual Palpation of the Gall-bladder	103
9.	Diagram to Show Parts which must be Excised in a Case of Malignant Tumor at the Hepatico-cystic Juncture	125
10.	Resection of Choledochus, Closure of both Ends, and Cholecysto-duodenostomy	125
11.	Drainage of Choledochus after Resection	126
12.	Choledocho-enterostomy	126
13.	Diagram of Hepato-cholangeio-enterostomy	127
14.	Voelcker's Method of Choledocho-duodenostomy	132
15.	Aneurism of the Hepatic Artery	141
16.	Corset Liver	158
17.	Carcinoma of the Gall-bladder	228
18.	Rupture of the Liver	239
19.	Pancreatic Calculi	268
20.	Diagram to explain the Modern Theory of Diabetes	272
21.	Wide Opening of the Gastro-colic Omentum to Explore the Pancreas	304
22.	Lymph-nodes of the Bile-ducts and Head of the Pancreas	318
23.	Cholecysto-duodenostomy, Autopsy Specimen	322
24.	The "Pancreatic Point" of Desjardins and the "Pancreatico-Hepatic Area" of Chauffard and Rivet	334
25.	Mobilization of the Duodenum to Expose the Head of the Pancreas and Choledochus	344
26.	Pancreatostomy by the Method of Link	349
27.	Obstructive Jaundice, with Enlargement of the Liver and Gall-bladder, from Pancreatic Carcinoma	358
28.	Cyst of Pancreas Presenting beneath the Gastro-colic Omentum	376
29.	Cyst of Pancreas Presenting beneath the Gastro-hepatic Omentum	376
30.	Cyst of Pancreas growing into the Transverse Meso-colon	377
31.	Specimen of Pancreatic Cyst Removed by Excision	380
32.	Incisions for Operations on the Biliary Tract	443
33.	Abdominal Incisions	443
34.	Rotation of the Liver to Expose the Gall-bladder and Bile-ducts	446
35.	Cholecystostomy	449

Chapter		Page
36.	Cholecystostomy for Prolonged Drainage	453
37.	Cholecystostomy for very Prolonged Drainage	453
38.	Cholecystectomy: Cystic Duct Isolated and Clamped	455
39.	Cholecystectomy: Dissection of the Gall-bladder from its Hepatic Attachments	456
40.	Transduodenal Choledochotomy	465
41.	Retro-duodenal Choledochotomy: Exposure of Calculus	467
42.	Retro-duodenal Choledochotomy: Removal of Calculus	467
43.	Retro-duodenal choledochotomy: Probing the Hepatic Duct	468
44.	Retro-duodenal Choledochotomy: Drainage of Choledochus	468
45.	Cholecystenterostomy by means of a Murphy Button	470
46.	Abscess of the Liver, Showing Method of Approach across the Costophrenic sinus	472
47.	Transpleural Operation for Hepatic Abscess	473
48.	Transpleural Operation for Hepatic Abscess	473
49.	Suture of the Liver	476
50.	Incision for Total Cephalic Pancreatectomy	478
51.	Pancreato-enterostomy	479
52.	Pancreato-enterostomy	480

SURGERY OF THE UPPER ABDOMEN.

CHAPTER I.

SURGICAL DISEASES OF THE BILIARY TRACT.

General Ætiological, Pathological and Diagnostic Considerations.

The prime factor in all non-neoplastic surgical diseases of the biliary tract is some form of micro-organismal invasion. The consequences of this may be slight or grave, transient or chronic, accompanied by very slight or very decided structural changes, the formation of concretions, etc.; but invariably, we believe, the underlying, exciting cause has been infection of the tract by bacterial life. The only exception to this statement may be found in those rare instances where surgical diseases result from the catarrhal condition of the bile-passages consequent upon the action of some toxic substance, such as is seen in acute phosphorus poisoning.

Our present knowledge of the bacteriology of the biliary tract is the result of the work of numerous investigators. Many points still remain undecided, but enough has been definitely settled by investigations through experiments upon the lower animals, or at autopsy, or by observations at the operating table, to allow certain definite statements to be made, based upon fact rather than theory.[1] It is known that when bacteria gain entrance to the biliary tract they may give rise to infections that result (1) in a mild catarrhal condition of the tract; (2) in acute suppurative cholangeitis with or without empyema of the gall-bladder; (3) in mild, or severe, gangrenous cholecystitis; (4) in gall-stone forma-

[1] Free use has been made, in what follows, of the Mütter Lecture of the College of Physicians of Philadelphia (1905), by Dr. A. O. J. Kelly, late Pathologist to the German Hospital.

tion, etc. It is known that the bile itself is a medium for the growth of many forms of bacteria, and that it is not, as was formerly believed, a bactericidal agent.

Bacteriology of the Biliary Passages.—It generally has been claimed that normal bile in normal bile-passages is sterile. This view, according to Hoppe-Seyler, is supported by the researches of Mieczkowski who, when operating for diseases other than those of the biliary tract, examined the bile of fifteen normal gall-bladders and found it sterile in every instance. Lippman on the other hand demonstrated bacterial life in apparently normal common ducts and in the normal ampulla of Vater. Moreover, the researches of Bond, quoted subsequently (page 4), prove conclusively that it is possible to find bacteria in normal mucous ducts.

A bacteriological study of the biliary tract in 413 cases operated upon by the senior author at the German Hospital, made by Dr. A. O. J. Kelly, demonstrated the presence of bacteria in 184 cases, while the cultures remained sterile in 229, or 55.4 per cent. of the cases examined. Superficial consideration of these findings might lead one to claim that less than half of the conditions found at operation were due to bacteria. Such deductions, however, would be erroneous. A large majority of the operations performed on the biliary tract are for the ultimate, rather than the immediate consequences of infection. In operations for acute disease bacteria are present, with very few exceptions. Between the time of bacterial invasion and the time of operation, however, the biliary tract will clear itself of bacteria in about half of the cases.

According to Funke, the research work of Gilbert and Fournier and also of Chauffard shows that sterile bile may be obtained from patients in whom bacteria-bearing calculi are found, the bile having become sterile after the stones had formed. Funke also quotes Frankel and Krause as having made culture tests of bile at 128 autopsies with 105 sterile results; whereas Letienne found bacteria in twenty-four out of forty-two necropsy examinations.

The bacterium most frequently found in the biliary tract is the Bacillus coli communis, being followed in the order of frequency by the Bacillus typhosus.

The findings in 413 cases examined at the German Hospital were as follows:

	Cases.	Per Cent.
Bacillus coli communis was found in	125	30.2
Bacillus typhosus was found in	29	7.0
Staphylococcus pyogenes aureus was found in	7	1.7
Streptococcus pyogenes was found in	1	0.2
Staphylococcus pyogenes albus was found in	5	1.2
Bacillus pyocyaneus was found in	2	0.5
Bacillus coli communis and Streptococcus pyogenes aureus were found in	2	0.5
Unidentified bacilli were found in	13	3.1
The cultures remained sterile in	229	55.4

Among other bacteria that have been found in cultures made from bile may be mentioned the Bacillus subtilis, the Bacillus capsulatus aërogenes, the Bacillus limbatus, the Bacillus gasoformans, the Bacillus lactis erythrogenes, the Bacillus of Friedländer, the Bacillus choleræ, the Bacterium oxygenes, Leptothrix, etc.

Avenues of Infection.—Bacteria gain entrance to the biliary tract, in most instances, through the *portal circulation*, or through the duodenal opening of the *common bile-duct*. Invasion also takes place through the *general circulation*, through the *lymphatic circulation*, and possibly through the *walls of the biliary tract*. Of these various avenues of entrance the portal circulation is the most frequently traversed, although many investigators have claimed that the ductal opening in the duodenum is the chief gateway of entrance. It must be remembered that the duodenum, in a normal condition and especially when free from food, is almost free of germ life. Kelly points out two other important factors that must be considered in connection with the entrance of germs through the ductal opening. One is the sphincteric action of the ampulla of Vater, which Archibald, in recent experiments, has found to exert a force equivalent to about 600 mm. of water pressure within the duct,[1] the other is the peculiar flushing of the duct and its opening by the intermittent gushing of bile into the duodenum. Were there a constant, free opening of the ampulla, with a slow steady flow of bile through the opening, this entrance would be a more common avenue of infection than it is. During the presence of food in this portion of the intestine, which is practically the only time germs are present in the normal duodenum, there is an intermittent expulsion of bile (see Vol. I, p. 38)

[1] According to Naunyn, Oggi found it equal to 700 mm. of water pressure.

which naturally clears the opening and the first part of the duodenum of all bacteria that may become lodged there.

The comparative freedom of the pancreas from infectious diseases has been advanced as a strong argument against invasion taking place through the ampulla of Vater. The bacteria make no selection between the pancreatic and the common bile-ducts when they reach the crossroads where these two channels join, but those entering the pancreatic duct are confronted by much greater obstacles to overcome than their fellows who enter the bile channel. Many claim that the pancreatic juice is antagonistic to germ life, while the bile, especially when stagnant, stimulates instead of hinders the growth of many forms of bacteria. There is no such pool of stagnant pancreatic juice as there is of bile in the gall-bladder, and hence no suitable lodging-place where bacteria may thrive and multiply.

Bacteria have, however, been found in the normal diverticulum and in the lower end of the normal common duct. Lippman's investigations of normal biliary tracts prove conclusively that bacteria may be present in apparently normal tracts, he having found them in the middle and upper third of the common duct. The experimental work of Bond, moreover, shows that it is possible for micro-organisms to travel against the natural currents in mucous ducts. According to these researches, there is either a "back-current," which will carry particles of matter against the normal current, or there is some action of the component parts of the duct or its secretion which will have the same effect. Bond points out that this is not a mere power of capillary attraction, since life is necessary for the action. Further, the walls of the duct must be more or less in apposition and must be lined with mucus. Bond was able to recover through a fistula into the gall-bladder granules of indigo blue which had been administered by mouth. The experiments of Bond, with the results obtained by him, seem to prove conclusively that it is perfectly possible for bacteria to enter the common duct from the duodenum and traverse its length. This may be accomplished not only by the motile bacteria, such as the colon, typhoid, and paratyphoid bacilli, but also by the non-motile staphylococci and streptococci.

In diseased conditions of the duodenum or of the lower end of the common bile-duct, invasion by bacteria may take place much

more readily. Catarrhal conditions of the duodenum will cause œdema of the mouth of the ampulla of Vater and thus interfere greatly with the cleansing power of the bile, which is secreted under very low pressure, and which becomes more and more sluggish in its flow as the result of very slight obstruction. There is also an extension of the catarrhal condition from the duodenum through the diverticulum and along the common bile-duct.

The most probable pathway of entrance in the majority of cases of infection of the biliary tract is through the portal circulation. Adami, according to Kelly, has shown that under normal conditions bacteria may be found in the deeper layers of the intestine, in the portal circulation, and in the liver. He advances the theory that the leukocytes carry the bacteria to the lymphatic radicles and also to the radicles of the portal vein.[1] They may also be taken up by the thoracic duct and emptied into the general circulation. The natural resistive and bactericidal powers of the liver cells are sufficient to overcome and destroy most of the germs arriving through the portal circulation. If there should be a weakened condition of the hepatic cells, however, either through disease or overwork, many of the bacteria will escape destruction and be excreted with the bile. The researches of Lartigau definitely settled the fact that infection of the biliary tract may be a descending one from the liver. Having tied the common duct in animals, Lartigau fed them bacteria, especially the Bacillus pyocyaneus, and was able to recover this germ from the gall-bladder in almost 50 per cent. of the animals so treated. In many instances the Bacillus pyocyaneus was recovered in pure cultures; in other instances the colon bacillus was associated with it. Bishop holds similar views, and thinks the portal vein the most frequent channel for entrance of bacteria to the bile-passages.

Infection of the biliary tract through the systemic circulation is possible and, in many instances, probable. The advancement in the study of the blood which has made it possible to determine the presence of bacteria in the general circulation in certain infectious diseases, such as typhoid fever, has made the systemic circulation seem much more important as a pathway of entrance for bacteria to the biliary tract. The work of Dörr proves that

[1] It is in this way that *appendicitis* and other infections in the abdomen may act as a predisposing factor for infections of the biliary tract.

bacteria injected into the circulation of a rabbit, can be recovered from the gall-bladder in a few hours. As pointed out by Kelly the infections of the biliary tract so often found as complications of systemic infections also show the possibility and probability of infection through the blood. It cannot be stated definitely, however, whether the infection, in these cases, takes place through the hepatic and cystic arteries, through the portal vein, or through the common duct from the intestinal canal. The presence of typhoid bacilli, unassociated with other bacteria, in the gall-bladder in cases of enteric fever naturally strengthens the belief that in some instances at least the systemic circulation conveys the germ to the biliary tract. This belief is confirmed by the experiments and findings of Joseph Koch (see page 19).

Wrzosek, as quoted by Else, claims that the bacteria are picked up by the lacteals and then are carried to the general circulation through the thoracic duct. Two series of experiments made by Wrzosek convinced him that bacteria are carried to the biliary tract by way of the general circulation through the thoracic duct much more frequently than they are through the portal vein. Else's two series of experiments, rabbits and dogs being the animals used, convinced him of the correctness of the following conclusions: "1. Infection usually reaches the gall-bladder by ascending the common and cystic ducts from the bowel. 2. Infection readily reaches the gall-bladder through the cystic artery. 3. Infection rarely or never reaches the gall-bladder by passing directly from the portal vein to the bile radicals."

In exceptional cases infection of the biliary tract may take place through the lymphatic system, as was probably the case in an instance reported by Müller. Bishop claims that infection may take place through this channel, although its occurrence is very rare.

Infection through the walls of the biliary tract, and especially through the walls of the gall-bladder, may occur by *contiguity*. It is questionable, however, whether the infection travels from without inward in those cases where the gall-bladder is surrounded by a mass of adhesions; it is much more likely that they are relics of a former inflammatory condition which probably had its origin within the bile passages. Under such circumstances any new

inflammatory processes are more likely to be a relighting of the old infection rather than a new one.

Pathogenesis.—After the micro-organisms have entered the biliary tract the consequences of their activity will vary with the virulence of the invading bacteria, the presence or absence of calculi, the resistance offered by the structures affected, and the anatomical peculiarities of the portion of the tract involved. These changes may vary from a very mild, quickly subsiding acute infection, to the most violent, fulminating, gangrenous process; from a mild, almost symptomless catarrh to a chronic inflammation of the biliary tract which may last for years, with acute exacerbations causing greater structural changes with each attack. Or the tract, and especially the gall-bladder, may be utilized as a store-house for masses of bacteria, as frequently demonstrated in the cases of "chronic typhoid carriers" (see page 26).

In many instances the only changes that can be noted are a slight swelling of the mucous membrane, with possibly some slight exfoliation of the epithelium. Such cases usually rid themselves of the infection through the natural resistance of the individual, and by an increase in the flow of bile which washes the germs from the tract, thus allowing the affected area to heal with only trifling change of structure. With infection of greater virulence, or where lessened resistance exists, an acute inflammation of the ducts or gall-bladder or both, with or without pus formation, will be found. Infection of the gall-bladder is more persistent than that of the ducts on account of the anatomical difficulties the gall-bladder has in freeing itself of the infectious material. The gall-bladder may be considered a diverticulum of the common duct, connected with the latter by a narrow channel, the cystic duct. There is a distinct S-shaped curve, sometimes called the antrum, where the gall-bladder narrows to become continuous with the cystic duct, and a spiral valve exists in the cystic duct, owing to the disposition of its mucous folds. The fundus of the gall-bladder usually is lower than the opening of the cystic duct. These factors greatly influence the drainage of the gall-bladder through the natural channels.

The presence of inflammation in the ducts or gall-bladder will cause the mucous membrane lining the cystic duct to become swollen with consequent narrowing of its lumen and damming of

bile behind the obstruction. The partial or complete stasis of bile thus produced affords germs an opportunity to exert their full power, the result being determined by the virulence of the micro-organisms present, and the ability of the gall-bladder and ducts to resist and overcome the infection.

Gall-stone Formation.

Historical.—Many and various theories have been advanced at different times to account for the formation of gall-stones. A more exact knowledge of the anatomy of the biliary tract, with a better understanding of the physiological action of the liver and the physiology and chemistry of the bile, enhanced as these have been by the pathological findings at the operating table, have enabled modern scientists to master the most important parts of the subject. The work of Naunyn settled the question definitely in the opinion of his followers. The researches of Kramer, of Bacmeister, and of Bishop have, however, thrown doubt on some of Naunyn's findings. A few questions concerning gall-stone formation remain unanswered, although they are not of great importance. The entire subject has been carefully reviewed recently by Exner and Heyrovsky.

Originally, gall-stones were supposed to be formed from coagulated bile, the coagulation being caused by a rise in the temperature of the liver. Paracelsus, according to Quincke, advanced the theory that gastro-intestinal disturbances caused an acid condition of the blood, the acids which were formed acting upon the bile with consequent precipitation and formation of concretions. Later, as a better knowledge of the normal constituents of bile was obtained, it was thought that a super-abundance of any of its various elements might exist, with consequent stone formation. Meckel called attention to a chronic catarrhal condition of the mucous membrane of the bile-tract and especially of the gall-bladder, and advanced the theory that a "stone-forming catarrh" might be the basis of the lithiasis. Flint advanced the theory that excessive brain work caused a condition of cholesteræmia; this gave rise to the theory that gall-stones were formed as soon as an excessive amount of cholesterin was present in the blood. Hein thought that plugs of mucus, the product of a diseased con-

dition of the mucous membrane, acted as nuclei around which the stones were formed. He also claimed that changes taking place in the bile within the bile-tract might cause the formation of stones—small, minute, bile-pigment stones first being formed in the ducts; and later, after they had reached the gall-bladder, acting as nuclei for larger stones. The supposition that the epithelial cells of the gall-bladder were important factors in gall-stone formation was suggested by Seifert. Naunyn advanced the idea that most stones were formed within the gall-bladder as a result of a catarrhal condition of the mucous membrane consequent upon bacterial invasion, desquamation of epithelial cells, and deposition from these cells of larger quantities of cholesterin than the bile could hold in solution. He also claimed that the calcium bile-salts acted as a cement substance, through the action of which accretions were constantly added to the original nucleus or stone. Naunyn also thought that the bile-pigment stones were formed in the duct through oxidation of bile, this being brought about by the action of bacteria. Naunyn's belief is generally accepted to-day, although later investigators have differed with him as to the origin of the excess of cholesterin.

Pathogenesis.—The actual formation of gall-stones is caused by an increase of the *cholesterin* constituent of the bile, a deposition of *calcium bile-salts*, especially bilirubin-calcium, in the presence of a *stasis or sluggishness in flow of bile.* The increase in cholesterin and the deposition of the calcium salts are due to the action of bacteria. This has been shown by the experiments of Gérard and of Bacmeister. Neither of these observers was able to produce concretions in sterile bile contained in a test-tube, but when colon bacilli were present deposition of salts readily occurred. The interference with the natural flow of bile also may be due to the action of bacteria in creating a catarrhal inflammation of the mucous membrane of the gall-bladder and ducts; or it may be consequent upon any of those predisposing causes of cholelithiasis (see page 88) which have a tendency to cause stagnation of bile. It is necessary for the inflammatory changes caused by the bacteria to be of a very mild character; the bacteria themselves must be almost non-virulent. A virulent inflammation of the gall-bladder or bile-tracts will cause rapid changes in the structures involved, acute cholecystitis with its train of compli-

cations and sequels (see page 50), a cholangeitis of purulent character, etc., rather than the mild, almost symptomless inflammation that is necessary for gall-stone formation.

The source of the increased amount of *cholesterin* is a question that has not been definitely settled. Naunyn attributes it to the cells of the mucous membrane lining the biliary tract which are exfoliated in excessive numbers as a consequence of a very mild, catarrhal inflammation. These cells contain droplets of myelin, of fluid cholesterin. Kramer on the other hand, has derived results from test-tube experiments which make it very probable that the cholesterin may be deposited from the bile itself. He was able to obtain a precipitate from a culture medium (consisting of equal parts of bouillon and bile) which had been inoculated with either the colon bacillus or the typhoid bacillus. The precipitate increased with the age of the culture. Microscopically the precipitate consisted of biliary coloring matter, cholesterin, magnesium phosphate, calcium phosphate, calcium carbonate, and bacilli. Kramer concluded from these experiments that "gall-stone formation is due to a chemical decomposition of bile, the direct result of the growth of micro-organisms therein." Bacmeister also differs from Naunyn regarding the source of cholesterin, and from Kramer as to the direct cause of its deposition from bile. He claims that there is a more or less constant autolysis of the bile, with the deposition of cholesterin from it; that this autolysis is much more marked when the bile is contaminated by epithelial cells or bacteria. Experimentally, he was able to demonstrate the separation of cholesterin crystals from sterile bile, and even from filtered bile, although the separation in the latter instance was much slower than in unfiltered bile. In the presence of the Bacillus pyocyaneus, the Bacillus proteus, the Bacillus typhosus, or the Bacillus coli communis he was able to demonstrate a very marked precipitation of cholesterin. Bishop holds that an inflammatory condition of the mucous membrane of the biliary tract is the initial step in gall-stone formation, the change in the membrane being the direct result of bacterial invasion, the bacteria penetrating the walls of the tract with the resultant mild inflammation. The result of this inflammation is an outpouring of cholesterin in larger quantities than can be dissolved by the bile. This view

is in keeping with that of Naunyn and is well summarized by Kelly who says that "the increased cholesterin, then, is derived not from the bile," "it results from catarrhal disintegration of the mucous cells lining the wall of the gall-bladder" (the seat of the formation of most gall-stones).

All observers are agreed that the mild inflammation of the mucous membrane causes an increased secretion of mucus, an albuminous substance which causes precipitation of the *calcium bile-salts*, especially the bilirubin-calcium. There is also an increase in the exfoliation of epithelial cells. Naunyn claims that the mucus, cholesterin and epithelial cells act as a nucleus for the beginning stone, being held together by the bilirubin-calcium which acts as a cement substance. Bacmeister, on the contrary, claims that the first stone formed is composed, almost invariably, of pure cholesterin; that the presence of this stone creates conditions very favorable for bacterial infection with inflammatory changes in the walls of the gall-bladder, and that these changes cause marked increase in the secretion of mucus. This mucus is very rich in calcium salts which furnish material for the formation of new stones, as well as for the deposition of additional layers on the surface of the stone already formed. Basing their views largely on the experimental work of Bacmeister, Aschoff and Bacmeister, in their recent monograph on Cholelithiasis, claim that with very few exceptions the first calculus—a cholesterin stone with radially disposed crystals—is formed without any bacterial infection of the biliary tract, merely by chemical changes in the stagnant bile of a gall-bladder more or less obstructed by mechanical means, such as kinks of the cystic duct, a viscid state of the bile, etc.

The *sluggishness in the flow of bile*, or even actual *stasis* is favored by two principal factors: (1) changes in the bile itself; (2) changes in the bile-ducts. The composition of the bile is altered by the chemical changes already alluded to, induced by bacteria; it becomes thicker, more viscid, less fluid. The bile-ducts become narrowed and eventually obstructed by inflammatory œdema, by kinks due to the drag of an inflamed gall-bladder, by impaction of calculi, or by pressure of surrounding adhesions, tumors, etc. When a calculus once becomes impacted, the bile

behind it is dammed up, and the formation of further calculi is favored.

Finally, a word should be said about calculous formation around *foreign bodies* as a nucleus (page 90). Langenbuch refers to several cases where concretions formed in the gall-bladder or ducts around intestinal parasites, needles, prunestones, and other foreign bodies, some of which entered through cholecysto-gastric fistulæ and others through the ampulla of Vater. Homans observed several stones which formed around silk ligatures introduced at an operation done seventeen months previously. Kehr reported three similar cases. According to Moynihan the experimental introduction by Meyer of sterile ivory balls into the gall-bladders of dogs failed to induce the formation of calculi; whereas in the test-tube experiments of Mignot, where attenuated growths of micro-organisms were first made, the subsequent introduction of foreign bodies resulted in the deposition of cholesterin on their surface. These experiments are in accord with the belief that bacterial infection is the underlying cause of gall-stone formation.

The *rapidity of gall-stone formation* is much greater than commonly supposed. Naunyn, in 1905, stated his belief that they might be formed within a few hours. Aschoff and Bacmeister claim, however, that the radial cholesterin stone is of extremely slow formation, many years being required to produce one of nut-size.

Composition of Gall-stones.—Chemically considered, gall-stones consist principally of cholesterin, various compounds of calcium with the different bile-pigments, and calcium carbonate. Among other chemical substances at times found in gall-stones may be mentioned phosphoric acid, bile acids, calcium sulphate, free bilirubin, copper, manganese, salicylic acid, etc. The chief bile-pigment found in combination with calcium is bilirubin. The most frequent chemical constituents of biliary calculi are cholesterin, calcium carbonate, and bilirubin calcium. These three, with few exceptions, are found in every stone, in varying proportions, combined with mucus and a nitrogenous compound which is supposed to result from the epithelial cells destroyed during the formation of the concretion (Hoppe-Seyler). The proportion of the various constituents of gall-stones varies considerably, the general characteristics of the stone varying, also, with the amount of each constituent present.

Physical Characters of Gall-stones.—The *color* depends upon the composition of the stone, varying from the almost pure white or yellowish stone, which is composed of cholesterin, to the black stone which contains an abundance of bile-coloring matter. The color varies in all stones, even the practically pure cholesterin stone showing dark patches of coloring matter between the crystals, and no stone being uniform throughout. The surface is generally darker than the inside of the stone; while the nucleus is, as a rule, lighter in color than the surrounding parts.

The *hardness* of the stone varies in proportion to the amount of calcium salts present. The cholesterin stones, while more or less firm and hard to the touch, are brittle and can readily be broken between the thumb and fingers. Stones recently formed generally are soft.

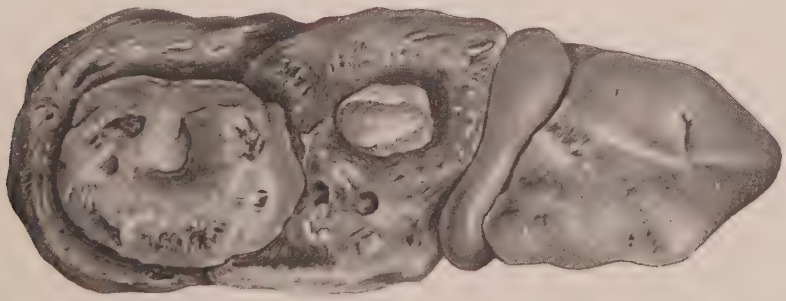

Fig. 1.—Mass of Gall-stones Conglomerated and Pressed into the Shape of the Gall-bladder. From a Patient in the German Hospital.

The *shape* of the concretions generally varies with the number present. Single stones are more or less rounded in shape, and of course, never faceted. When a number of stones are present, they are usually faceted, being pressed into this shape by the contractions of the gall-bladder while they are still soft. As a rule, a large mass of stones, unless greatly disturbed, will take the form of the gall-bladder, the portions of the stones on the outside of the mass and in contact with the walls of the gall-bladder being usually rough and more or less rounded, while the portion of each stone in contact with other stones will be pressed flat. The stones in the center of the mass are almost universally faceted.

Naunyn asserts that this faceting is due entirely to pressure, and not to a grinding motion among the stones; but it is difficult to see how some of the facets become so highly polished unless it is by attrition.

The "mulberry" stones generally are composed of a great number of small concretions which have coalesced, finally being covered with a new layer which gives the stones their appearance of uniformity.

In *weight* and *size* gall-stones vary from almost imperceptible particles to concretions 6 inches long. The senior author has seen them so small that it was impossible to detect their presence through the walls of the gall-bladder, although they were present in hundreds. Packard mentions the following as being noteworthy among the larger stones reported: "Richter records one weighing three ounces and five drams removed from the common duct at autopsy.[1] Schüppel states that he has seen one measuring 7.5 cm. in length, 4 cm. in width, and 12 cm. in circumference, and refers to one (reported by Meckel) which measured 15 cm. by 6 cm. Russell reports one measuring $5\frac{3}{4}$ by $4\frac{1}{2}$ inches. Thornton removed from the common duct during life a stone 2 inches long and $3\frac{1}{2}$ inches in circumference. Frerichs says that he has seen a number of stones measuring from 2 to $2\frac{1}{2}$ inches by 1 inch. It may be roughly stated that the size of the stones as a rule varies inversely with their number." W. Bartlett, of St. Louis, has successfully removed from the common duct a stone measuring 4 by $1\frac{1}{2}$ inches, and weighing two ounces and a half.

The *number* of stones present in any given case may vary from one to thousands. There seems to have been nothing discovered which will definitely account for their variation in number. The number formed may depend upon the amount of calcium salts present. These act as a cement substance; and when they are present in abundance, there may be a coalescence of a greater number of the particles of mucus, cholesterin and epithelial cells which generally form the nuclei, and a correspondingly smaller number of fully developed calculi; while with a deficiency in the calcium salts, the particles may remain distinct and separate, thus presenting a much greater number of nuclei. The largest

[1] Rolleston claims this to be the largest stone ever described. The case was published in 1793.

number of calculi found in any one case, according to Rolleston, was recorded by Otto, who counted 7802.

It is probable that the stones usually originate in the gall-bladder, and that they are all formed nearly simultaneously; but when a solitary stone becomes impacted in one of the ducts, we see no good reason for disputing the possibility of the subsequent formation of other stones in the stagnated bile, provided the factors necessary for gall-stone formation are still present.

According to Rolleston, Mignot concluded from experimental researches that additional calculi might be formed in recurrent attacks of cholecystitis. The teaching of Aschoff and Bacmeister, that the "radial cholesterin stone" is always formed first, without bacterial infection, and that subsequent bacterial invasion causes the deposition of calcium bile-stones, has already been referred to (page 11). Naunyn, as quoted by Kelly, after drawing attention to the fact that where numerous calculi are found, they are almost always alike in general appearance, comes to the conclusion that gall-stones in a single individual always are formed from a single infection of the gall-bladder, and that recurrences of such infection in that particular individual are very rare. Kehr thinks that the vast majority of stones are formed in the gall-bladder and that those instances of stone formation taking place within the ducts are "extremely rare."

Gall-stones may be **classified** in many ways. Most authors follow Naunyn, who describes six varieties:

1. *Pure Cholesterin Stones.*—These are more or less uncommon, although this variety is the one that Aschoff and Bacmeister claim precedes the formation of all other varieties. As a rule they are of large size. They generally present the following characteristics: they are oval, at times being spheroid; they are very hard, but brittle, so that they may be crushed between the thumb and finger; the surface may be either smooth or nodular; in color they vary from either pure white or yellowish to brownish-black on the surface, but are white and generally crystalline in the interior. They are not stratified, the cholesterin crystals being radially arranged around a comparatively soft center of amorphous material.

2. *A laminated variety of cholesterin stones.*—These are composed of cholesterin, bilirubin-calcium, biliverdin-calcium, and cal-

cium carbonate. In these calculi there is usually about 90 per cent. of cholesterin. They may occur singly or in varying numbers, when they are at times faceted. A section reveals laminations, layers of almost pure white cholesterin alternating with layers which may be yellowish, brown, red, or green in color. The body of the stone generally is *non-crystalline*, while the center or nucleus may be crystalline or may be hollow when the calculus has become dry. These calculi, according to Aschoff and Bacmeister, usually are formed by accretions around the primary "radial cholesterin stone" as a nucleus.

3. *The Common Gall-stones, or Mixed Cholesterin Calculi.*—They vary greatly in number and in size and usually are faceted. The surface varies in color from yellow, the usual color, to a brown or at times, a white color. Their consistency depends a great deal upon the time of their examination. When freshly removed from the gall-bladder they may be rather soft with a "soapy" feel; after they have dried out they become harder. The center of this variety is also, at times, a cavity; it usually is softer than the outside.

4. *Mixed bilirubin-calcium stones,* in which there is a preponderance of the bile-pigment salt, with about 25 per cent. of cholesterin. They are often of considerable size. They generally occur singly or in groups of threes or fours, in which instances they may be faceted. On section they show concentric layers of dark material which is generally reddish-brown in color. The interior of the stones as a rule is rather soft; on drying, the stones usually contract with the formation of fissures.

5. *Pure bilirubin-calcium stones,* which are of two types: They may be small brownish-black calculi, with irregular surfaces, and showing a tendency to adhere one to the other; this type is rather soft in consistency. The other type is much larger and much smoother on the surface, which usually exhibits a metallic luster, while the interior of the stone is rather spongy in consistency.

6. In this class Naunyn places some *rare forms* including among them calcareous stones, pearly bodies which consist of amorphous or partly crystalline cholesterin, casts of the biliary passages, etc.

The pathology, symptomatology, diagnosis, and treatment of

gall-stone disease will be considered in a subsequent chapter (see page 64).

REFERENCES.

Ætiology and Pathology.

Archibald: The Canadian Med. Assoc. Jour., July, 1912.
Aschoff and Bacmeister: Die Cholelithiasis, Jena, 1909.
Bacmeister: Münch. med. Woch., 1908, lv, 211, 283, 339, 915.
Bartlett: Report of Trans. West. Surg. and Gyn. Assoc., in N. Y. Med. Jour., 1908, i, 326.
Bishop: Brit. Med. Jour., 1907, i, 676.
Bond: Brit Med. Jour., 1905, ii, 232.
Dörr: Wien. klin. Woch. 1905, xviii, 884; Centralbl. f. Bakteriol. (Originale), 1905, xxxix, 624.
Else: Surg., Gyn., and Obst., 1910, ii, 470.
Exner and Heyrovsky: Arch. f. klin. Chir., 1908, lxxxvi, 609.
Funke: N. Y. Med. Jour., 1906, ii, 1077.
Gérard: Comptes Rendus de la Soc. de Biol. de Paris, 1905, lviii, 348.
Homans: Annals of Surg., 1897, xxvi, 114.
Hoppe-Seyler: Nothnagel's Encylopædia, Diseases of Liver, Pancreas, etc., Philadelphia, 1903, p. 525.
Kehr: Gall-stone Disease, translated by W. W. Seymour, Phila., 1901.
Kelly: Amer. Jour. Med. Sc., 1906, ii, 446, 744.
Kelly: Osler's Modern Medicine, Phila., 1908, vol. v, p. 691.
Koch: Centralbl. f. Bakteriol., 1908, xlii, Beiheft, S. 54.
Kramer: Jour. of Exper. Med., 1907, ix, 319.
Langenbuch: Chir. der Leber u. Gallenblase, Stuttgart, 1897, ii, 195.
Lartigau: California State Jour. of Med., 1906, iv, 17.
Lippman: Le Microbisme biliaire normal et pathologique, Paris, 1904, p. 75.
Moynihan: Gall-stones and their Surgical Treatment, Phila., 1905, p. 19.
Müller: Brooklyn Med. Jour., 1905, xix, 11.
Naunyn: Klinik der Cholelithiasis. Leipzig, 1892.
Naunyn: Mitth. a. d. Grenzgeb. d. Med. u. Chir., 1905, xiv, 537.
Packard: Nothnagel's Encyclopædia, Diseases of Liver, Pancreas, etc., Phila., 1903, p. 533.
Quincke: Nothnagel's Encyclopædia, Diseases of Liver, Pancreas, etc., Phila., 1903, p. 469.
Rolleston: Diseases of the Liver, Gall-bladder and Bile-ducts, Phila., 1905.

Typhoid Infections of the Biliary Tract.

The infections of the biliary tract by the typhoid and paratyphoid bacilli are becoming daily of greater importance. Not only because of the surgical conditions that arise from such infec-

tions is this true, but also from the standpoint of public health and hygiene. Many epidemics of enteric fever have been traced directly to persons who were suffering from a more or less remote typhoid infection of the gall-bladder and bile-ducts. The usual medical measures, such as cholagogues, are of no value in ridding the biliary tract of the Bacillus typhosus. The vaccine treatment may prove of great value but its efficiency has not been established; in the case recently reported by Leary it was entirely useless. It has already become necessary for the surgeon to devise ways and means for accomplishing the end in view—ridding the system of of the remaining typhoid bacilli. The treatment of these cases is considered at page 26.

Infections of the gall-bladder and bile-tracts have been recognized as complications of typhoid fever by almost every one who has studied many cases of the latter affection. Since 1897, when Mason reviewed the history of this complication of typhoid, numerous investigators have carefully studied the subject and, as Kelly says, have reached a comparatively unanimous conclusion "(1) that the typhoid bacillus is regularly present in the gall-bladder, and commonly in pure culture, in practically all cases of typhoid fever—indeed, it is the one region of the body from which a pure culture of the organism is most likely to be obtained; (2) that the typhoid bacillus may persist in the gall-bladder, as well as within gall-stones, weeks, months, even years after the patient has recovered from an attack of typhoid fever; (3) that cholangitis and cholecystitis (catarrhal, suppurative, and gangrenous) are by no means infrequent complications of typhoid fever; and (4) that a history of antecedent typhoid fever may be obtained in many cholelithic and cholecystic subjects."

The presence of the bacilli in the gall-bladder is explained in two ways. According to the generally accepted theory, the germs are carried to the liver by the portal circulation (see page 5). The normal action of the liver cells is bactericidal. It is the generally accepted theory, as explained at page 5, that bacteria are constantly passing from the intestinal tract to the liver, in the portal circulation. Adami thinks they must be transported by the leukocytes. Normally these bacteria are destroyed in the liver; but if the liver cells become overburdened by the work thrown upon them, or are poisoned by the toxins thrown out by the

bacilli, their bactericidal power is lost, and live bacilli are then excreted with the bile. That infection may occur also by the systemic circulation is suggested by the observations of Joseph Koch, Wrzosek, and Else. Koch discovered inflammatory infiltration in the mucous membrane in a case of beginning typhoid cholecystitis, the infiltrate containing dense clumps of bacilli resembling capillary emboli. From these foci, he claims, the bacilli pass through the epithelium into the interior of the gall-bladder. Koch also reports having followed the transit of the bacilli from the capillary emboli in the submucosa through the walls of the gall-bladder in experimental work on rabbits. In these experiments he injected typhoid bacilli into the veins of rabbits and recovered them from the gall-bladder even in those cases in which he had previously tied the cystic duct.

Although the excretion (or secretion) of bile by the liver cells is almost constant, its discharge into the duodenum varies with the needs of the digestive functions. During the periods of digestive quiescence the bile backs up through the cystic duct into the gall-bladder where it remains until needed in the intestinal tract (Vol. I, page 38). Unfortunately for typhoid fever patients, as far as the biliary tract is concerned, they are kept on an extremely low diet, large quantities of bile not being required for its digestion and absorption. In consequence of this fact, the gall-bladder naturally becomes distended with bile, and the latter becomes more or less loaded with mucus. If the mucus causes a partial obstruction of the cystic duct, the bile will become stagnated and this will allow rapid multiplication of any bacteria that may be present. Under these conditions, with good resistance on the part of the patient, there will result a slight inflammation in the gall-bladder, so slight in fact that it usually is not mentioned by the patient nor recognized by the physician as a complication of the typhoid. This condition is overlooked in many instances because the patient's sensibilities are numbed by the typhoid infection and he does not experience sufficient pain to attract the attention of the physician; and the physician will overlook it, unless, as Kelly points out, "systematic and repeated examinations of the gall-bladder region are undertaken."

With lessened resistance, the bacilli are allowed greater freedom of action, and there results a more pronounced cholecystitis, with

or without ulceration of the mucous membrane or even of the entire wall of the gall-bladder, and, at times, perforation of that viscus.

In the presence of pre-existing cholelithiasis, infection of the gall-bladder during the course of typhoid fever might be expected to cause marked inflammatory changes, due to the fact that pathological lesions already exist in that viscus as a result of the presence of the calculi.

No satisfactory statistics as to the frequency with which the gall-bladder is involved in typhoid fever have been published. The junior author in analyzing the reports of the Episcopal Hospital found that among 2864 cases of typhoid fever there were but eighteen or 0.62 per cent. in which infection of the gall-bladder was recorded as a complication. Thomas, in a series of 895 cases of typhoid, found cholecystitis in twelve or 1.3 per cent.

In connection with two cases of perforation of the gall-bladder during typhoid fever treated by operation, the junior author analyzed the reports of nineteen other operations on the gall-bladder during typhoid fever. The lesions found at operation were as follows:

	Cases.	Recovered.	Died.	Mortality Per Cent.
Cholecystitis alone	4	2	2	50.00
Cholecystitis and empyema of the gall-bladder	3	0	3	100.00
Cholecystitis, empyema, peritonitis	4	2	2	50.00
Perforation with peritonitis	6	4	2	33.30
Perforation (found only at autopsy)	4	0	4	100.00
Total	21	8	13	61.90

Quénu collected forty-five operations for lesions of the gall-bladder during or soon after an attack of typhoid fever.

Thomas has analyzed 154 cases of typhoidal cholecystitis collected from the literature. He reports that perforation of the gall-bladder occurred in thirty-nine or 25.3 per cent. Eleven of these patients were operated upon with a mortality of 54.6 per cent. The remaining twenty-eight died without operation. The Bacillus typhosus was isolated in about 50 per cent. of the operated cases; calculi were found in three. In the series reported by the junior

author the typhoid bacillus was recovered in pure culture from the gall-bladders of nine patients or 42.8 per cent. In one case, the typhoid bacillus was associated with the colon bacillus; in another case the paracolon bacillus was found; in ten cases the bacteriological findings were not recorded.

Symptoms and Treatment of Gall-bladder Disease during Typhoid Fever.—Ashhurst points out that there are two quite distinct classes of cases: In the first there is a more or less gradual onset of abdominal pain, fairly well localized (by patients who are conscious) to the right hypochondrium, accompanied by localized tenderness, and frequently by a papable mass easily recognized as

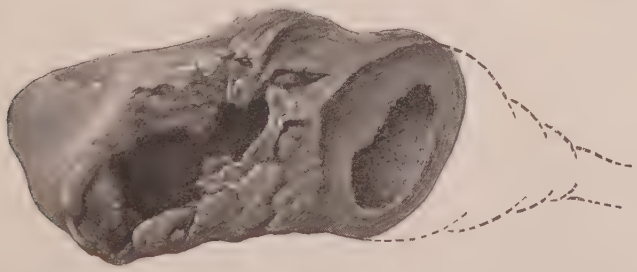

FIG. 2.—PERFORATION OF THE GALL-BLADDER DURING TYPHOID FEVER; CHOLECYSTECTOMY; RECOVERY. NATURAL SIZE. (*Ashhurst.*)

the distended gall-bladder. If operation is undertaken at this stage, there is found (*a*) cholecystitis; (*b*) empyema; or (*c*) empyema and commencing suppurative pericholecystitis. In the second class of cases the symptoms already mentioned have existed for a period varying from a few hours to a week or ten days, when suddenly there is an acute attack of abdominal pain, accompanied by a fall of temperature (noted in eight out of ten cases), and sometimes by sweating; these signs being gradually followed, when immediate operation is not undertaken, by a secondary rise of temperature, a spread of the pain and tenderness over the whole abdomen, and increase of distention as the inital rigidity disappears.

When cholecystitis is recognized as a complication during an attack of typhoid fever, it is not desirable to resort immediately to operation. The condition should be closely watched, and only when it is thought that empyema is present or perforation threatens, should the gall-bladder be drained. If perforation occurs, no delay should be allowed; in most cases cholecystectomy

is the operation of choice. If the cholecystitis subsides without operation, the patient should be kept under observation, and such dietetic and hygienic treatment should be instituted as is suitable in cases of cholelithiasis, since the subsequent development of calculi is very probable.

Sequels of Typhoid Infection of the Biliary Tract.—It is almost impossible to arrive at definite conclusions in regard to the sequels of typhoid infections of the biliary tract. In a series of 521 operations upon the gall-bladder and bile-ducts for cholelithiasis performed by the senior author at the German Hospital a positive history of antecedent typhoid was obtained from 127 or 24.3 per cent. The time elapsing between the attacks of typhoid and the time of operation varied from two weeks to forty-one years. Camac found that among thirty-three cases the time elapsed since the onset of the typhoid fever varied from ten days to one year or more. It is questionable whether all of these cases should be classed as sequels of typhoid fever, and there is no definite method known by which this question can be decided. If the decision is based solely on the bacteriological findings, one conclusion will be reached; if, however, the pathology of gall-bladder infections is remembered, and especially the fact that the gall-bladder may rid itself of all bacteria after the subsidence of the acute infection, a different conclusion may be reached. Among a series of eighty-one patients, at the German Hospital, who gave a history of antecedent typhoid infection, the contents of the gall-bladder were examined bacteriologically in forty-six; of these, thirteen cases, or 28.4 per cent., gave cultures of the bacillus typhosus. It cannot, however, be stated positively that none of the remaining sixty-eight cases, which also had an antecedent history of typhoid, should be classed as sequels of typhoid fever; since these might be cases in which the gall-bladder had rid itself of the Bacillus typhosus after this infection had caused structural changes, the deposition of concretions, etc. In fifteen or 32.6 per cent. of the forty-six examinations, pure cultures of the colon bacillus were obtained. It is uncertain whether these cases should be classed as sequels of the antecedent typhoid, or as cholelithiasis resulting from the invasion of the gall-bladder by the colon bacillus; but our belief is that the calculous formation in these cases, with possibly a few exceptions, was

a direct result of the pathological changes consequent upon the typhoid infection. There might also be classed in the same category seventeen (36.9 per cent.) of the forty-six cases in which the gall-bladder was sterile at the time of operation, and one case in which Staphylococcus aureus was found.

A general summary of the thirteen cases giving a pure culture of the Bacillus typhosus follows:

Sex.—All were females. *Age.*—The age varied from twenty-three to fifty-six years, at time of operation, the average age of the thirteen being 40.3 years. *Time since convalescence from typhoid:* This varied from two weeks to forty-one years, the time being respectively, two weeks; one month; five weeks; four months; five months; nine months; three years; seven years; eight years; fourteen years; fifteen years; forty-one years; one case not stated. *Operations:* Cholecystectomy was performed upon six cases; cholecystectomy with choledochostomy, upon three; cholecystostomy upon three; choledochostomy upon one. *Results:* All recovered.

Thomas in his analysis of 154 cases, quoted above, which he classes as complications or sequels of typhoid, found the Bacillus typhosus in 50 per cent.; but in those patients in whom the gall-bladder condition arose during the course of typhoid fever, the typhoid bacillus was present in the gall-bladder in perhaps 95 per cent.

More definite findings will have to be obtained before cases similar to those in the various series mentioned above can be classed with certainty as complications or sequels of typhoid, and not acute or chronic diseases of the biliary tract in patients in whom an antecedent attack of typhoid fever was but a coincidence, although we believe that most of these cases should be placed in the former category; certainly when the typhoid bacillus is found in the gall-bladder the presumption is warranted that it has caused the disease.

Another very interesting condition has been made evident by routine bacteriological examinations made in cases of operation on the gall-bladder. This is the fact that *injection of the biliary tract by the Bacillus typhosus may occur in patients who have never presented any symptoms of typhoid fever*, the invasion giving rise to a primary typhoid cholecystitis. Quénu points out that typhoid

fever is not primarily an intestinal disease (wherefore the term *enteric* fever is to be avoided), but a primary septicæmia; and that the liver is the primary agent for the elimination of the Bacillus typhosus, which is brought to it by the blood. In this manner the bile becomes infected; and he asserts that many jaundices—hepatic, epidemic, or simple febrile in character—are nothing less than primary infections of the bile passages by the bacillus typhosus, and he thinks Weil's disease is produced in the same way. Further study, we think, is required before the assertions of Quénu can be endorsed without qualification; but it seems altogether probable that such studies, particularly in respect to blood cultures, will in the near future prove the truth of his contentions, and afford a scientific explanation for the occurrence of these rare cases. The following seven patients operated upon by the senior author at the German Hospital, belong to this class.

1. K. H., female, aged forty-nine years, married. Has had eleven pregnancies. Has never had typhoid fever. Ten years before admission to the German Hospital, June 22, 1903, had severe pain in epigastrium; recurrent attacks of pain had been experienced about twice a year since then. Operation revealed a thickened and contracted gall-bladder with extensive adhesions between the gall-bladder, duodenum, and stomach, and a fistula between the stomach and gall-bladder. Gall-stones were present in the mass of adhesions and in the gall-bladder. Cholecystectomy and gastrorrhaphy were performed, the patient recovering. The Bacillus typhosus was obtained from the gall-bladder.

2. A. C., female, aged forty-seven years, admitted to the German Hospital April 27, 1904. Has never had typhoid fever. Present trouble began two years before admission, with epigastric pains which lasted a couple of days. Has had frequent recurrences of pain, a very severe attack occurring three months before admission, lasting twenty-four hours, and being followed by marked tenderness over the gall-bladder region, and icterus which persisted for about four days. Operation revealed pericholecystic adhesions, an enlarged gall-bladder containing about 100 cc. of bloody, yellowish fluid, and many stones. Cholecystectomy was performed. The patient recovered. Pure culture of Bacillus typhosus was obtained from the gall-bladder.

3. E. H., female, aged twenty-seven, married, no pregnancies. Has never had typhoid fever. Has had several attacks of epigastric pain, radiating to right side and right shoulder. Last attack three weeks before admission to German Hospital, March 11, 1904. The attack of pain was followed by a chill accompanied by fever and vomiting. Markedly tender over gall-bladder region. Operation revealed adhesions between the gall-bladder,

liver, duodenum and omentum. The gall-bladder was enlarged and contained about 100 cc. of pus, with one calculus obstructing the cystic duct. Cholecystectomy was performed, the patient recovering. Cultures of the Bacillus typhosus were obtained from the gall-bladder.

4. J. W. male, aged twenty-five, admitted to the German Hospital June 12, 1907. Has never had typhoid fever. Has always been well with exception of an attack of influenza in May, 1906. Three days before admission had an attack of severe, cramp-like abdominal pains, centering in the epigastrium; no nausea or vomiting. Skin had a yellowish tint; there was marked tenderness over the gall-bladder region. Operation revealed an enlarged gall-bladder covered with recent exudate; 50 cc. of pus removed by canula. Gall-bladder walls markedly thickened and inflamed. The viscus contained, besides pus, three calculi, each the size of a nutmeg, and numerous small, yellowish faceted stones. One large stone was located in the neck of the gall-bladder obstructing the cystic duct. Cholecystostomy was performed, the patient recovering. Cultures of Bacillus typhosus were obtained from the gall-bladder.

5. M. D., female, aged thirty-six, married. Admitted to the German Hospital October 25, 1907. Has had five pregnancies. Uses alcohol freely. Has never had typhoid fever. One year before admission had first attack of severe pain in upper abdomen, followed by chills and fever, and jaundice. Pains were referred to right arm. A tender, sore point was found between the ribs and umbilicus. No mass palpable. Operation revealed adhesions between the gall-bladder and stomach. The gall-bladder was small with thick walls and full of yellowish, faceted calculi. Cholecystectomy was performed, the patient recovering. Pure cultures of the Bacillus typhosus were obtained from the gall-bladder.

6. E. Z., female, aged twenty-three, admitted to the German Hospital December 4, 1907. Has had one pregnancy. Has never had typhoid fever. One year before admission had first attack of epigastric pain, which was sudden in onset, very severe, and referred to the right scapula. No chills or fever. Has had attacks similar to the first one every week or two since. On admission her skin was icteric, and the gall-bladder region was very tender. Operation revealed "cob-web" adhesions between the gall-bladder, liver and stomach. The gall-bladder was markedly contracted. Two stones were in the common duct. A choledochostomy was performed, the patient recovering. Cultures of Bacillus typhosus were obtained from the common duct.

7. F. R., male, aged fifty-eight. Admitted to the German Hospital December 24, 1906. Has never had typhoid fever or other acute infectious disease. Present trouble began about sixteen years ago with pain in the epigastrium. One year ago had severe pains, colicky in character, in gall-bladder region, with chills, nausea, and vomiting. Was jaundiced. Has marked jaundice with decided tenderness over the gall-bladder region. Operation revealed adhesions between gall-bladder and omentum; gall-bladder enlarged, chronically inflamed and pouched. The viscus was filled with calculi which obstructed the cystic duct. Cholecystectomy was performed,

the patient recovering. Pure cultures of Bacillus typhosus were obtained from the gall-bladder.

Typhoid Carriers.—From the standpoint of public health, the most important cases of typhoid infections of the biliary tract are found in those patients who become chronic "*typhoid carriers.*" In this condition the patient recovers from the attack of typhoid but does not rid himself of the typhoid bacilli, which remain, supposedly in the gall-bladder, being discharged from that viscus into the intestinal tract and excreted with the fæces. Repeated instances of epidemics arising from such a source have been brought to the attention of the medical profession, infection generally occurring through the milk supply or through food prepared by such a typhoid carrier. The proportion of chronic carriers has been estimated at from 1.7 per cent. to as high as 5 per cent. of the total number of typhoid cases. The bacilli have been found in the livers of these patients at autopsy, as well as in the walls of the gall-bladder and in the bile (Hammond). The typhoid bacilli may remain in the gall-bladder for an indefinite period. Gregg is quoted by Kelly as having recovered the bacillus fifty-two years after the subsidence of the acute attack; and Jundell reports twenty-two cases of infection extending over a period of fifty-four years, arising from a "carrier" who herself had never had an attack of typhoid fever.

The diagnosis of "chronic carrier" is not made from any symptoms referable to the gall-bladder or gall-ducts. Usually the patients are perfectly normal symptomatically, so far as the liver and biliary tract are concerned. The only positive means of diagnosis is finding the bacilli in the stools of the suspected "carrier," or in the stomach contents after a special test-meal as advised by Weber. Following the procedure suggested by Volhard, Weber administers 200 c.c. of olive oil by mouth when the stomach is empty. After an interval of half an hour the stomach contents are withdrawn. The oil is supposed to induce a copious flow of pancreatic juice, some of which will find its way into the stomach, carrying with it bile. Weber claims that, if bile be recovered from the stomach contents, the bacillus of typhoid will be found in every patient who is a typhoid carrier.

The best treatment for such cases it is difficult to decide, because sufficient data have not been collected definitely to deter-

mine whether any given form of treatment will suffice in all cases. Forster claims that the bacilli will disappear from the stools when the existing lesion in the gall-bladder has healed. To accomplish this he advises the administration of cholagogues, preferably bile or some preparation of bile; if that fails, drainage of the gall-bladder is indicated. Grimme favors operation, and reports a case in which the gall-bladder, containing thirty-five small calculi, was removed with disappearance of the bacilli from the stools. Probably the first instance of an operation done solely for the purpose of ridding the gall-bladder of the bacillii of typhoid in a "chronic carrier" was performed by Dehler. His patient was an insane woman who had infected a number of other women. The gall-bladder gave rise to no symptoms, but the Bacillus typhosus was found in the stools in thirty-seven out of thirty-nine examinations. At the operation it was found that there were adhesions surrounding the gall-bladder; the viscus contained two calculi, one of which partially obstructed the cystic duct. Cholecystostomy was performed. The operation was followed by complete absence of typhoid bacilli from the stools. We agree with Dehler who thinks operative interference should be instituted in all cases of "chronic carriers" where the desired result cannot be obtained by the use of serotherapy or other means. Leary has recently reported two operations for the condition of typhoid carrier, both entirely successful after conservative treatment had failed. In most cases cholecystectomy is the operation of choice.

REFERENCES.

Ashhurst, A. P. C.: Amer. Jour. Med. Sc., 1908, i, 541.
Camac: Johns Hopkins Hosp. Repts., 1900, viii, 339.
Dehler: Münch. med Woch., 1907, i, 779.
Editorial: Jour. Amer. Med. Assoc., 1908, i, 1906.
Forster: Münch. med. Woch., 1908, i, 1.
Grimme: Ibid., loc. cit., S. 16.
Hammond: Jour. Amer. Med. Assoc., 1909, i, 48.
Jundell: Hygiea, Festband, 1908, in Jour. Amer. Med. Assoc., 1909, i, 388.
Kelly: Amer. Jour. Med. Sciences, 1906, ii, 455.
Kelly: Osler's Modern Med., 1908, v, 818.
Koch: Centralbl. f. Bakteriol., 1908, xlii, Beiheft, S. 54.
Leary: Jour. Amer. Med. Assoc., 1913, i, 1293.
Mason: Trans. Assoc. Amer. Phys. 1897, xii, 23.

Quénu: Revue de Chir., 1908, xxxvii, 828.
Soper: Jour. Amer. Med. Assoc., 1907, i, 2019.
Thomas, B. A.: N. Y. Med. Jour., 1907, ii, 688.
Weber: Münch. Med. Woch., 1908, ii, 2443.

Jaundice as a Symptom.

Jaundice as a sign or symptom is of importance to the surgeon whenever it is present in any patient who comes under his care. The term is meant to signify a peculiar discoloration of the tissues of the body by bile-pigment. It is not a pathological entity, but a symptom of a number of lesions that are found in connection with diseases of the liver and bile-passages, or the structures adjacent to them. These lesions that are of interest in the surgery of the liver and bile-tracts must be recognized and dissociated from those which are of medical significance only.

In a healthy individual, bile-pigment is found only in the bile. Under normal conditions these pigments are formed in the liver, as has been determined by the experiments of Naunyn and Minkowski, as quoted by Quincke. After ligation of the common duct in birds, biliverdin was found in the urine after about one and one-half hours, and in the blood after five hours; after extirpation of the liver this pigment could not be obtained from the blood and was present in the urine only in traces. Whether it is true or not that icterus cannot exist without participation of the liver in the process, as is claimed by Quincke and others, it may be stated that the liver or its excretory ducts are involved clinically, with very rare, if any, exceptions, in every case of jaundice seen by the physician or surgeon. It is essential for the diagnostician to differentiate between those cases requiring surgical treatment and those that will be relieved by medical measures alone.

In the majority of cases jaundice results from absorption of bile from the small radicles of the bile-ducts, due to obstruction of these channels. When it is realized that the secretion-pressure of the bile amounts to only 200 mm. of water, as determined by Heidenhain in experiments made on guinea-pigs and dogs, it is evident that only slight obstruction to the flow of bile is necessary to overcome the secretion-pressure. When obstruction exists in the bile-passages, either intrahepatic or extrahepatic, there is a damming up of the bile in the proximal portions of the ducts

extending as far as the capillary radicles. As the secretion of bile continues, there naturally will follow increased dilatation of the ducts. The capillaries gradually become lengthened and distended until they rupture into the adjoining lymph-spaces. The bile-pigments are then absorbed by the lymphatics, and are carried to the thoracic duct and into the general circulation. That the bile-pigments are carried to the blood through the thoracic duct has been repeatedly demonstrated, according to Kelly. After the common bile-duct was ligated bile-pigments were soon recovered through a fistula in the thoracic duct; when both thoracic and common ducts were ligated, thus preventing the admission of bile into the general circulation through the thoracic duct, discoloration of the skin was seldom seen, and the bile-pigments did not appear in the urine for several days. It is also possible for the bile-pigments to enter the systemic circulation through the intrahepatic capillaries (Gerhardt; Queirolo and Benvenute).

There are numerous instances of icterus occurring in patients where it is impossible to demonstrate an obstruction to the bile-ducts. In such cases the manner of absorption of the bile-pigments is not so plain. But it is probable that the mechanics are in every case much as described above, although various theories have been advanced to account for the occurrence of jaundice when no obstruction is evident. According to Kelly, Eppinger believes that obstruction takes place even in these instances, but only in the most minute radicles of the bile-channels: stasis of bile occurs and there is dilatation of the biliary canaliculi with final rupture into the pericellular lymph-spaces.

In some instances of injury to the liver or gall-ducts with escape of free bile into the peritoneal cavity, jaundice may follow its absorption. If there is a chronic peritonitis present, however, the peritoneum will be incapable of absorbing the bile-pigments, and jaundice may not occur.

The presence of bile-pigments in the circulating blood gives rise to certain **symptoms** which are entirely independent of the primary cause of the condition. These are most marked in obstructive jaundice, and vary somewhat in degree with the completeness of the obstruction. The most noticeable symptom produced is discoloration of the skin and visible mucous membranes. In the onset of icterus the color is generally a light

yellow, gradually changing to a greenish tint, and terminating in a yellowish-gray. The natural pigmentation of the skin will often mask the lighter degrees of jaundice, so that it is possibly, even probably, often overlooked in brunettes. The scleræ, as a rule, are discolored more rapidly than the skin.

Itching of the skin is more or less prominent as a symptom of icterus, often preceding the discoloration, although as a rule it follows the latter condition. There is no relation between the intensity of the discoloration of the skin and the degree of pruritus.

Douglas states that xanthoma (xanthelasma) may occur in chronic lithiasis with jaundice. This skin lesion is not peculiar to jaundice although often seen in connection with it. It consists of pale yellowish spots, found chiefly in the region of the eyelids, the areas of discoloration being raised above the level of the surrounding skin. According to Quincke the yellowish pigmentation of these spots is not caused by bile-pigments, nor has their connection with icterus been definitely decided.

The symptom of jaundice is frequently observed first on the visible mucous membranes, especially the scleral conjunctivæ. This change is not always apparent on the other mucous membranes except on the lids and hard palate, where the membrane is naturally more or less pale. It usually can be demonstrated on the visible mucous membranes, however, by depriving them of their blood temporarily by pressure. The discoloration is thus often made manifest by pressing a thin glass slide against the everted lower lip. As a rule the discoloration of jaundice is more marked in the regions of natural pigmentation, and may often be apparent on the abdomen when not noticeable in the conjunctivæ.

Because of the excretion of the greater part of the bile-pigments through the kidneys, the urine is discolored, the color depending to a great extent upon the presence of the different pigments and their products. The color varies from yellowish, through yellowish-red, to a greenish-brown. This discoloration of the urine is generally seen before the pigmentation of the skin, preceding the latter at times by several days. The presence of bile-pigments in the urine is in reality the first clinical symptom of jaundice. It can be demonstrated by the discoloration of the

froth after a brisk shaking of the urine; and filter-paper through which the urine is passed usually will be stained by the pigments. The more delicate tests are given in text-books of medicine and in laboratory manuals.

In protracted cases of jaundice, the urine may be diminished in amount, with increase in the specific gravity. Hyaline casts, at times bile-stained, are found. Douglas claims that bile does not appear in the urine in the absence of jaundice; he also thinks that scanty urine with marked jaundice is a precursor of death.

Hemorrhage in Cases of Jaundice.—The action or effect of the bile-pigments on the blood has not been definitely determined. They are present in the blood in every case of jaundice. There follows, generally, and especially in cases of long-standing jaundice, an impoverished condition of the blood with a diminution in the number of erythrocytes and in the amount of hæmoglobin. There is also present a condition that is of the greatest importance to the surgeon, a decided prolongation in the clotting time of the blood. The coagulation period will be increased from the normal, three or four minutes, to as much as fifteen minutes and even longer. It has not been determined whether this condition is consequent upon a change in the fibrin ferment, upon alterations that occur in the red blood-cells, or due to the mere presence of the bile-pigments and bile-acids. Schwarz and Ottenberg claim that the tendency to hemorrhage is independent of the degree of icterus and that it may be evident after all jaundice has disappeared. They believe that it is not the mere presence of bile in the blood that causes the tendency, but the presence of a toxic substance of unknown nature, which acts by preventing the secretion of thrombo-kinase. This activating substance of coagulation never is present in the circulating, blood but is produced from nucleated cells, chiefly blood-platelets. Lloyd speaks of this condition as a pseudohæmophilia. When it is present there may be bleeding from the mouth, the nose, the intestines, the bronchi, the kidneys, and into the skin. There is not infrequently an oozing from the wound which will continue until the patient is exsanguinated. The oozing may be absolutely uncontrollable. This bleeding may not begin at the time of the incision; the wound may be dry when closed and a feeling of safety from hemorrhage may be thus engendered, only to find that the dressings are soaked with blood two, three or even four days after

the operation, by an uncontrollable oozing that will continue until the death of the patient. This danger cannot be prevented, but it may be lessened somewhat by the administration of calcium chloride in 30-grain doses every three or four hours for a day or more before the operation, by mouth, and 60-grain doses after the operation, by rectum. Adrenalin, gelatin, and other drugs have been tried without avail. Calcium chloride aids the coagulability of the blood but it does not always suffice to prevent the oozing. Recent studies made by Addis seem to prove that very slight effect is obtained by the doses of calcium chloride usually administered. The use of thyroid extract is occasionally of benefit in such cases.

Mayo says "when serious hemorrhage occurs we find that packing the wound with a two-inch strip of iodoform gauze, incorporating in the packing a considerable quantity of a mixture of one part acetanilid and seven parts boric acid used dry, is the most effective procedure."

Serum therapy has given better results in these cases than any other treatment, although it is not entirely satisfactory nor is its use without danger. Rabbit, horse, guinea-pig and human serum have been used with varying success. Crile and others strongly advocate direct blood transfusion.

The main objections to the use of serum are the production of "serum sickness," well described in 1905 by von Pirquet and Schick in their monograph on that subject; and the production of anaphylaxis. Welch describes "serum sickness" as a condition characterized by irregular fevers, followed by migrating skin rashes and troublesome urticaria, local œdema, itching which may become agonizing, painful lymph-nodes near the site of injection; the joints may be swollen, painful and red; the urine may contain albumen, blood, and casts; and there may be hemorrhage from the mucous membranes.

In absolute anaphylaxis there is sudden dyspnœa, cyanosis and death.

The use of rabbit serum is preferred by Leary because it is easily obtained and there is no danger of producing tetanus which might result from the use of horse-serum. He claims that the danger of "serum sickness" and of anaphylaxis is lessened by using a different serum if a second dose is required. Unpleasant re-

sults are seldom seen if all doses of the serum are administered within a period of ten or twelve days. Besredka claims that anaphylaxis can be prevented by injecting the serum in fractional doses.

Welch advises the use of human serum and describes a special apparatus which facilitates its recovery from human blood. The dangers of "serum sickness" and of anaphylaxis are lessened when human serum is used.

Schwarz and Ottenberg conclude from their studies that "the expectation that the injection of serum will alter the coagulating power of the circulating blood runs so contrary to our physiological data, that we prefer to remain skeptical as to the value of this form of treatment."

Bernheim has made a number of laboratory tests to influence rapid coagulation of the blood, using an extract made from blood-vessel walls. He claims remarkable results.

As noted at page 226, Quénu uses antidiphtheritic serum, 20 cc. of which he injects on the day before the operation.

Our experience has been limited to the use of gelatin, adrenalin, calcium salts, and horse and human serum. None of these have yielded the results hoped for. In most cases where hemorrhage has been controlled, ultimately, we have felt that the result was due as much to the the administration of morphine and the careful, firm packing with gauze as to the use of any of the various remedies.

The serum may be administered subcutaneously, into the peritoneal cavity, into the spinal fluid, or directly into a vein. From 10 cc. to 30 cc. may be administered twice daily for 3 days subcutaneously and oftener if bleeding occurs.

The **absence of bile-pigments from the stools** will cause the excreta to be lighter in color than normal; the well-known "clay-colored stools" are often seen, or there may be simply a lighter color than normal. When the stools vary in color, showing alternately the presence and absence of bile-pigments, it may be concluded that the obstruction to the flow of bile is intermittent, a condition that is found in the so-called "ball-valve" obstruction of the common duct by a calculus. In obstruction due to a tumor, such as cancer of the head of the pancreas, the obstruction does not vary, and the stools remain constantly free from bile-pigments, and of a grayish color.

The color of the stools seen in jaundice is not due entirely to

the absence of bile-pigments; there is also an increase of the undigested fats which have a decided influence on the consistency and color of the stool. Kelly claims that this amount of imperfectly digested fat may increase from normal (7 to 10 per cent.) to as high as 80 per cent.

To comprehend the true significance of jaundice as a symptom in surgical diseases of the liver and biliary passages, we must consider the various causes which will give rise to it, and the frequency with which it is found in those diseases which are treated by the surgeon. Icterus occurs whenever there is a more or less prolonged obstruction of the hepatic or common duct, from any cause. It occurs, according to Kelly "under other circumstances—in which apparently the biliary ducts are patent, as in cirrhosis and other diffuse diseases of the liver; in many infections, such as the different types of so-called infectious jaundice, syphilis, yellow fever, septicopyæmia, malaria, pneumonia, typhoid fever, etc.; in intoxications such as poisoning with ptomains, phosphorus, arseniuretted hydrogen, chloroform, mushrooms, toluylenediamin, pyrogallol, snake venom, coal-tar products, etc.; in acute yellow atrophy of the liver; in progressive pernicious anæmia and hæmoglobinæmia; in disturbances of the circulation, such as passive congestion; in certain nervous perturbations (so-called emotional jaundice, menstrual jaundice, etc.); in the new-born, etc."

Hunter gives the following classification of jaundice not due to obstruction:

1. Jaundice produced by the action of poisons, such as phosphorus, arsenic, and snake venom.
2. Jaundice met with in various specific fevers and conditions, such as yellow fever, malaria (remittent and intermittent), pyæmia, relapsing fever, typhus, typhoid fever, and scarlatina.
3. Jaundice met with in various conditions of unknown or more or less obscure, infectious nature, and variously designated as "epidemic," "infectious," or "malignant" jaundice, "icterus gravis," "Weil's disease," and "acute yellow atrophy of the liver."

To these might be added the jaundice frequently associated with severe hemorrhage, with starvation, or with lowered blood-

pressure in the portal or hepatic vessels in the presence of increased tension in the smaller bile-ducts.

Jaundice does not occur in every case of biliary tract disease, whether it is catarrhal inflammation, calculus in the ducts, or other lesion. A history, carefully taken, will show that icterus has been present at some time in the course of the disease in about 65 per cent. of the cases, and has not been noted in about 35 per cent. At the time of operation, however, jaundice is present in only about 35 per cent. of the patients. These figures are not in accord with those of the Mayos who state that jaundice was present in 70 per cent. of their patients at the time of operation. The statistics of Kelly combined with those of Mencke, including an analysis of the histories of 606 patients who had been operated upon by the senior author at the German Hospital, give the following figures: Of 606 patients, 373 or 61.5 per cent. had jaundice at some time or other; 212 or 34.9 per cent. had never had jaundice; and in the case of twenty-one or 3.5 per cent. there was no statement as to the occurrence of jaundice. One-hundred and fifty-six or 25.7 per cent. had jaundice at the time of operation and also had had previous attacks of icterus; twenty-eight or 4.6 per cent. had jaundice at the time of operation, but had never had it previously; 183 or 30.2 per cent. had no jaundice at time of operation but had had jaundice at some previous time; six or 0.9 per cent. had jaundice at time of operation, but no statement was made as to the presence or absence of icterus at a previous time. The summary of these cases will show that 190 or 31.3 per cent. had jaundice at the time of operation; 395 or 65.1 per cent. had no jaundice at time of operation (in twenty-one or 3.4 per cent. the presence or absence of jaundice was not noted); 360 or 59.4 per cent. had had attacks of jaundice prior to operation.

Extra-hepatic jaundice, which is found in connection with those diseases or conditions which demand the aid of the surgeon for relief, is due to some form of obstruction to the passage of bile through the ducts. The following is a convenient classification:

1. Obstruction due to inflammatory thickening of the mucous membrane of the ducts, or the result of such inflammation; rarely to tumors of the ducts themselves.

2. Obstruction due to stone in the common, rarely the hepatic, duct; or in the cystic, pressing on the common duct.
3. Obstruction due to neoplastic or hyperplastic conditions of the pancreas, or its lymphatics, especially those of the head of this organ.
4. Obstruction due to neoplasms or to pathological conditions such as adhesions, enlarged lymph-nodes, kinks, etc., of neighboring organs exerting pressure on the common duct.

It is impossible to discover the true significance of icterus in every case. Jaundice is a symptom and not a disease; underlying every case there is a lesion to account for the presence of this symptom, but great difficulty is often encountered in determining just what this lesion is. A complete history and careful study of the accompanying symptoms will, in the majority of cases, make the diagnosis clear; in other cases an exploratory operation will be necessary.

Jaundice which is slight and persists most probably is independent of obstruction.

Jaundice from obstruction becomes intense very rapidly.

Jaundice coming on gradually but ultimately becoming intense, with clay-colored stools, generally is due to pressure from neighboring structures, especially to diseases of the pancreas, such as pancreatic lymphangeitis, chronic interstitial pancreatitis, or carcinoma of the pancreas.

Jaundice which does not persist indefinitely or which recurs time and again, generally is due to calculous obstruction.

Jaundice with sudden onset, accompanied by colicky pains and clay-colored stools, generally is due to obstruction within the gall-ducts.

Jaundice following severe paroxysms of pain generally is due to gall-stone formation or to carcinoma. In the latter case there should be a history of failing health before the onset of pain or jaundice.

Jaundice in the presence of an enlarged liver generally is due to cirrhosis of the liver, to cancer of the liver, or to pyæmic abscess of the liver.

Jaundice with ascites generally is due to cancer of the liver or to cirrhosis. In the former there are darting pains, loss of weight and intense jaundice. In cirrhosis there generally is a history of alcoholic dyspepsia; the jaundice is generally much less intense than in cancer.

Jaundice with pyrexia is secondary either to acute febrile infection, to suppurative pylephlebitis, or to inflammation of the bile-ducts. Temporary pyrexia may be caused by the passage of a stone through the bile-ducts.

Jaundice with a history of previous attacks generally is due to a catarrhal condition of the bile-ducts or to the presence of gall-stones.

Jaundice with cerebral symptoms generally is due to acute atrophy of the liver, to poisoning by phosphorus, or to some specific fever such as pneumonia.

Jaundice in a young person preceded by symptoms of gastric catarrh generally is "catarrhal jaundice" (page 45).

Jaundice which is intermittent, at times slight and again intense, with urine that varies in color from light to dark, with stools that are intermittently dark and clay-colored, with colicky pains, with chills and fever similar to those of malaria, almost invariably is due to chronic calculous obstruction of the common duct.

Jaundice with fatty stools, in the presence of glycosuria, is generally indicative of pancreatic disease.

Jaundice increasing, without remissions, with marked dyspeptic symptoms, with marked increase in neutral fats in the stools, with undigested muscle fibres with nuclei intact in the stools, with a positive Cammidge's test, with a positive Sahli's test, is due to pancreatic disease.

Jaundice following general failure of health, increasing until it becomes absolute and never varying, with a greenish tint, with rapid loss of weight, with a distended gall-bladder, with gradual and painless onset, generally is due to carcinoma of the head of the pancreas.

In jaundice due to obstruction, either internal or external, the stools are clay-colored or lighter in color than normal, either continuously or intermittently.

In jaundice not due to obstruction of the bile-ducts, the stools are not clay-colored. It must be remembered that certain drugs,

such as bismuth, iron, and charcoal will color the stools; altered, disintegrated blood in the stools will also change the color.

Jaundice is seen in non-calculous cholecystitis only after the inflammatory process in the gall-bladder has extended to the mucous membrane of the cystic and common ducts.

The significance of jaundice as a symptom in various diseases of the liver and biliary tract will be considered more fully in the sections devoted to those lesions.

REFERENCES.

Addis: Quarterly Jour. of Med., 1909, ii, 149.
Bernheim: Jour. Amer. Med. Assoc., 1910, ii, 283.
Besredka: Annales de l'Inst. Pasteur, 1910, xxiv, 879.
Deaver: N. Y. Med. Jour., 1903, ii, 301.
Deaver: Amer. Jour. Med. Sc., 1908, i, 37.
Douglas: Surg. Diseases of Abdomen, Phila., 1903, pp. 364 and 365.
Gerhardt: cited by Quincke, loc. cit., p. 427.
Heidenhain: cited by Quincke, loc. cit., p. 425.
Hunter: Allbutt's System of Medicine, London, 1897, v, p. 82.
Kelly, A. O. J.: Amer. Jour. Med. Sc., 1906, ii, 446; 744.
Kelly, J. A.: N. Y. Med. Jour., 1910, xci, 334.
Kelly, A. O. J.: Osler's Modern Med., Phila., 1908, v, p. 691.
Leary: Med. Commun. Mass. Med. Soc., 1908, xxi, 121.
Lloyd: N. Y. Med. Jour., 1906, ii, 222.
Queirolo and Benvenuti: cited by Quincke, loc. cit.
Quénu: Rev. de Chir., 1909, xxxix, 462.
Quincke: Nothnagel's Encyclopædia, Diseases of Liver, Pancreas, and Suprarenal Glands, Phila., 1903, p. 423.
Schwarz and Ottenberg: Amer. Jour. Med. Sc., 1910, ii, 26.
von Pirquet and Schick: Die Serumkrankheit, Leipzig u. Wien., 1905.
Welch: Amer. Jour. Med. Sc., 1910, i, 800.

CHAPTER II.

SURGERY OF THE GALL-BLADDER AND BILE-DUCTS.

Deformities, Anomalies and Malpositions.

Congenital Obliteration of the Bile-ducts.—The absence of a passageway for the bile, at birth, usually is the result of obliteration or atresia of the common bile-duct. Lavenson suggests that the condition is a true atresia, and not an "obliteration," the latter presupposing the existence of a lumen which subsequently becomes destroyed. The condition, although unusual, is not rare. Lavenson collected sixty-two cases from the literature.

Paul Matthieu collected eight authentic and twelve doubtful cases of **congenital stricture** of the bile-ducts; the stricture usually has been at or near the duodenal termination of the common duct.

Other rare anomalies include cases where there is more than one common duct, cases where one or more of the bile-ducts open into the stomach or at an unusual point in the intestine, and anomalies in the number and situation of the hepatic ducts.

Pathologically, there is an obstruction, atresia, or obliteration of the common duct, with cirrhosis of the liver. Lavenson thinks the cirrhosis is the result of the obliteration and consequent bile stasis; Rolleston supports the view that the cirrhosis precedes the obstruction, a descending cholangeitis causing obliteration of the lumen of the duct. As pointed out by Rolleston, other causes of stricture must be recognized, such as fœtal peritonitis, with its resulting adhesions; or obstruction of the ducts by syphilitic granulation tissue, as in a case under his own care.

The classification of these cases of infantile jaundice which has been adopted by Matthieu is convenient. He classifies them in two main divisions: (1) Those in which the condition is not compatible with life (congenital occlusion or total absence of the bile-duct); and (2) cases compatible with life, including both (*a*) congenital icterus without lesion of the bile-ducts (which may be attributed to angeiocholitis of the intrahepatic ducts or to splenic disease causing jaundice by hæmolysis), and (*b*) those cases already

mentioned, in which stricture, but not absolute occlusion of the ducts exists.

Clinically congenital obstruction of the common duct is characterized by jaundice which generally is present at birth, although it may not develop for some weeks. The jaundice increases in intensity, the urine contains bile and the stools are free from all bile-coloring matter. The liver and spleen generally are enlarged. Hemorrhage from the cord may occur, and purpuric spots may be seen. Emaciation rapidly ensues, with coma, stupor, and, at times, convulsions followed by death. One case is reported (Treves) where the patient lived nineteen years with continuous jaundice for sixteen years.

The *diagnosis* is not readily made. The condition may be distinguished from icterus neonatorum by the short duration of the latter, and the presence in that disease of bile in the stools and its absence from the urine (Griffith). In the jaundice of the new-born due to a simple catarrhal duodenitis or cholangeitis, there always is some degree of fever, which usually is absent in cases of icterus due to congenital anomaly or stricture. Partial occlusion of the duct causing jaundice which ultimately will clear up cannot be distinguished until time has proved the diagnosis. Hess uses a duodenal catheter as an aid in diagnosis. This is easily passed in infants up to two years of age. Being passed through the stomach and pylorus, the duodenal contents are pumped out, and are examined for bile, pancreatic juice, etc. In Hess's own patient life was preserved for three months, possibly because the accessory duct of the pancreas was patulous. Though tests made during life showed the presence of bile in the duodenal contents, complete occlusion of the common duct was found at autopsy; and Hess suggests that perhaps bile may be excreted from the circulation through the intestinal canal.

The *treatment* is operative, although antisyphilitic treatment should be instituted in the hope that the condition is due to congenital syphilis, even if the history of the patient is negative. The surgical procedure necessary would be some form of anastomosis between the upper, unobliterated portion of the duct, and the duodenum (page 124). Lavenson collected reports of four unsuccessful attempts at operative relief.

Congenital Absence of the Gall-bladder.—This anomaly of

development in man is a rare condition. Although the gall-bladder is not essential to life and is entirely absent in some of the lower animals, such as the rhinoceros, the camel, the goat, the deer, and the elephant, its congenital absence in man is rarely noted. Nearly all of the important information upon the subject, as pointed out by Stone, has been assembled by F. Fink and A. Bubenhofer. In most of the cases reported as such, as pointed out by Eshner, "the absence of the gall-bladder was associated with conditions that point to obliteration of a previously existing viscus, rather than to a condition of agenesis." Eshner reported the case of a child who came under his observation for a persistent cough. The bowels moved several times daily, the stools being pale. No jaundice was present. The patient died when two years of age, and at autopsy the liver was found to be of normal size and condition, although changes probably syphilitic in origin were apparent. The biliary vessels and ducts, with the exception of the cystic duct, were normal. The gall-bladder was absent, the usual fissure for the gall-bladder was wanting, and there was nothing suggestive of the gall-bladder ever having been present. Gay collected nineteen cases of absence of the gall-bladder; in six of them "absence" was the only abnormality. Stone's patient was a woman fifty-four years of age upon whom an operation was performed under the diagnosis of calculous cholecystitis, with obstruction of the common duct. A palpable mass was thought to be the gall-bladder filled with calculi. Upon opening the abdomen the gall-bladder was conspicuously absent, the duodenum and pylorus being attached to the normal site of that viscus. No adhesions were present. Calculi were readily palpated in the common and hepatic ducts. After the ducts had been opened and the stones removed, search for the gall-bladder was made but it could not be found. Exploration with a probe through the hepatic duct failed to reveal any vestige of the cystic duct. Niemack has recently reported a case of cholesterin stone in the common duct in a patient in whom he could not find a gall-bladder.

The condition does not present any sign or symptom that might lead to a correct diagnosis. The diagnosis is made either at the operating-table or postmortem. The *treatment* of the cases subjected to operation should be directed to the condition for which the operation is performed.

Anomalies in position and shape of the gall-bladder are not common. Constrictions, sacculations and other deviations from the normal shape of the viscus may be the result of inflammatory changes; the organ may become enormously distended as a result of obstruction of the cystic duct or may become practically obliterated by cicatricial contraction. Many of the cases of "hour-glass" gall-bladders that have been reported may be the result of a defect in development or the effect of cicatricial contraction. The gall-bladder may be bi-lobed, may be transversely placed, may be imbedded in the liver substance, or may be freely movable owing to the presence of a distinct mesentery. This condition is described as **wandering gall-bladder,** and is not exceptionally rare. Kübig notes having seen two cases at autopsy. Its importance arises from the fact that it predisposes to torsion or volvulus of the gall-bladder.

Sherren, who successfully removed at operation a **double gall-bladder,** refers to the autopsy reported by Purser as the only case where there existed two complete gall-bladders with separate cystic ducts; Purser himself quotes from the Philosophical Transactions (1693–94) a case where at postmortem there were found two distinct gall-bladders, one in the right lobe and the other in the left lobe of the liver.

Several cases of **bifid gall-bladder** are on record. The following case was observed at autopsy at the German Hospital.

Postmortem Record.—Mrs. H., aged fifty-five years, death from apoplexy. The gall-bladder was bi-lobed, the greater lobe being discolored and almost gangrenous at its lower portion. This lobe contained one calculus. The other lobe seemed to be normal. There was only one cystic duct, draining both lobes; it was patulous.

The following conditions found at operation by the senior author well illustrate some of these conditions.

Diverticulum of the Gall-bladder.—M. S., female, aged fifty-two. Operation, December 18, 1907. Peritoneum opened. Gall-bladder surrounded by omental adhesions which could be stripped off readily with a piece of gauze. The gall-bladder was distended and full of calculi of various sizes. The neck of the gall-bladder just above the cystic duct was pouched in such a way as to form a sac which pressed upon the common duct and caused obstructive jaundice. Cholecystectomy performed. Common duct did not contain calculi. Patient made an uneventful recovery and jaundice rapidly disappeared.

Malposition of Gall-bladder.—J. B., male, aged thirty-eight, admitted to German Hospital, October 27, 1905. Had had typhoid fever at eighteen. Was a heavy whiskey drinker. First attack of pain in gall-bladder region four years ago, followed by slight jaundice. Three weeks before admission had sharp pain followed by jaundice which became progressively deeper. Operation, October 30, 1905. Dense adhesions between gall-bladder, omentum, colon, liver, pylorus and abdominal wall. The gall-bladder was very deeply placed, "horizontally transverse." Calculi were found in the gall-bladder, three in the common duct, two in the ampulla of Vater, and one in the duct of Wirsung. Duodenum incised and stones removed from ampulla and pancreatic duct. The patient suddenly had failure of respiration and died in spite of energetic treatment.

Gall-bladder with a Mesentery.—G. W., male, aged fifty-seven. Present illness had lasted four years. Had had pain in gall-bladder region, referred to left epigastric region and to right spine; jaundice; chills and fever; nausea and vomiting. Operation, March 18, 1907. Gall-bladder found to be small and containing numerous calculi. The remains of pericholecystic inflammation were evident in numerous adhesions. The gall-bladder was freely movable after the adhesions were liberated, and had a distinct mesovesicæ which extended from near the fundus to the cystic duct. Cholecystectomy was performed. The common duct was explored but was found patulous and free of calculi. Patient made an uneventful recovery.

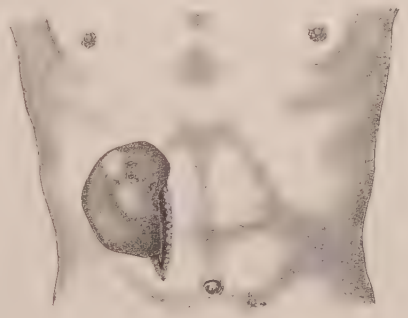

FIG. 3.—HOUR-GLASS GALL-BLADDER. (*From a patient in the German Hospital.*)

Hour-glass Gall-bladder.—A. S., female, aged thirty-five. Operation, May 24, 1904. Adhesions between the gall-bladder and stomach. These were ligated and cut and the gall-bladder was found to be "hour-glass" in shape, both pouches being filled with calculi (FIG. 3). Cholecystectomy was performed. Patient made a good recovery.

Hour-glass Gall-bladder.—W. S., female, age not stated. Operation, January 2, 1904. Adhesions between liver and duodenum. Gall-bladder was "hour-glass" in shape, the two portions being united by a fibrous band Distal portion, which was free from calculi, was removed. Proximal portion contained four stones. Cholecystostomy was performed. Patient made an uneventful recovery.

REFERENCES.

BUBENHOFER:[1] Anatom. Hefte, Wiesbaden, 1904–05, xxvii, 305.
ESHNER: Med. News, 1894, i, 548.

[1] This work has not been accessible to us.

Fink: Prag. med. Woch., 1904, xxix, 245.
Gay: Trans. Chicago Path. Soc., 1902, v, 108.
Griffith: Archives of Pediatrics, 1908, xxv, 174.
Kübig: Münch. med. Woch., 1912, lix, 1998.
Lavenson: Journ. of Med. Research, 1908, xviii, 61 (n. s. xiii).
Mathieu: Rev. de Chir., 1908, xxxvii, 61, 174.
Niemack: Surg., Gyn., and Obst., 1908, ii, 487.
Purser: Brit. Med. J., 1886, ii, 1102.
Rolleston: Brit. Med. J., 1907, ii, 947.
Sherren: Annals of Surg., 1911, liv, 204.
Stone: Amer. J. Med. Sc., 1908, i, 889.
Treves: quoted by Rolleston and Hayne, Brit. Med. J., 1901, i, 758.

Cholangeitis and Cholecystitis.

A thorough knowledge of the ætiology and pathology of the infectious diseases of the biliary tract is essential for every one who desires to interpret correctly the signs and symptoms presented. These vary greatly with the different pathological processes, and an accurate diagnosis can be made, the proper treatment instituted, and the ultimate outcome predicted, only through a correct interpretation of their significance. In some instances the lesions are varied and multiple, being diagnosticated correctly only after direct inspection and palpation. In the majorty of cases, however, a study of the signs and symptoms present will enable the surgeon to make a correct diagnosis.

All non-neoplastic surgical diseases of the biliary tract are the result of bacterial infection.[1] Infection of the biliary tract gives rise to inflammatory changes in its component parts, the mildness or severity of the lesion produced depending upon the virulence of the bacteria and the resistance of the patient. Mild or severe cholangeitis, mild or severe cholecystitis, with or without the formation of biliary calculi, will follow. At times the infection is so slight and so readily thrown off by the involved structures that no permanent pathological lesions result. In other instances the onset and course of the infection are very insidious, the resulting chronic inflammation, gall-stone formation, etc., not giving rise to important symptoms until many years after the true commencement of the disease.

[1] For a full description of the bacteria found in the biliary tract and their paths of entrance, see page 2.

Infections of the biliary ducts and gall-bladder usually coincide. In a few cases the infection of the gall-bladder may precede that of the ducts, but in the immense majority of cases the ducts are first affected; the infection in the ducts may subside while that in the gall-bladder continues; but infection of one without infection of the other is rare. The results of the infection, however, are very different; thus pathological lesions which result from infection of the gall-bladder without serious, or with quickly subsiding infection of the bile-ducts, will differ greatly from those seen when the bile-channels are permanently diseased. With involvement of the common and hepatic ducts, lesions of varying degrees of severity are always found in the liver; when the gall-bladder alone is infected, the liver is seldom involved. Infection of the ducts gives rise to hepatic and perihepatic lesions; infection of the gall-bladder without involvement of the ducts, gives rise to pericholecystic lesions.

Cholangeitis.—The simplest form of infection of the bile-ducts causes **acute catarrhal cholangeitis.** Usually this has the same ætiology as gastro-intestinal catarrh, which often is described by its most striking symptom, as "catarrhal jaundice." Cholangeitis frequently is a complication or sequel of this condition. It is also observed in connection with some of the infectious diseases, such as typhoid fever and pneumonia; it may result from the interference, by various toxins, with the normal activity of the ultimate radicles of the ducts in the liver, or with the metabolism of the liver cells; it may occur in cirrhosis of the liver, or in advanced heart disease.

Catarrhal jaundice is essentially obstructive jaundice. In the acute condition the duodenal mucous membrane around the bile-papilla, and that in the lower end of the common duct, become swollen and œdematous and cause partial obstruction of the biliary outlet; this is followed by jaundice of varying intensity. At times a plug of mucus is found in the diverticulum of Vater causing obstruction by its presence. Kelly (loc. cit., page 808) quotes Eppinger's findings in a case of catarrhal jaundice which came to necropsy as the result of an accident eight days after the onset of jaundice. Eppinger found hyperplasia of the lymphoid tissues of the mucous membrane in the portion of the common duct which transverses the intestinal wall. This had led

to complete obstruction of the duct and dilatation of the rest of the biliary system.

Mild jaundice, of the catarrhal type, may be of surgical significance in those cases where it is a complication or sequel of echinococcus cysts, gummata, carcinoma of the liver, etc., where jaundice results from obstruction caused by pressure on the ducts from without. Mayo Robson thinks that in many cases of catarrhal jaundice the head of the pancreas is inflamed and that the pressure of this upon the common bile-duct is sufficient to cause obstruction. These cases, however, usually are more properly classed as chronic catarrhal cholangeitis (page 47).

Acute catarrhal cholangeitis is of interest to the surgeon from a diagnostic standpoint. In the early stages, as a rule, the condition is hidden by the usual symptoms of gastro-intestinal catarrh, such as loss of appetite, coated tongue, foul breath., headache, nausea and vomiting. The temperature may rise to 100° F. or 101° F., or, in rare cases, it may be even higher, the patient also having rigors and sweats. Jaundice appears in from two to seven days, being first noticed in the sclera. With the onset of jaundice, as a rule, the temperature falls to normal or even subnormal. The stools become clay-colored, and the urine becomes scanty and high colored. The liver may be enlarged, but usually is not tender.

The *diagnosis* should be made from the age of the patient, the history of antecedent gastro-intestinal disturbance, the mildness of the attack, and the usual mild course which the disease pursues. Jaundice in those of middle life or in the aged is generally due to gall-stone disease, pancreatitis, or malignancy. Pain is more marked in these latter conditions, and usually is remittent in type; the jaundice varies in intensity unless there is obstruction of the common duct; and periodic attacks of chills and fever are much more common than in catarrhal jaundice.

The *prognosis* is good. Most cases of acute catarrhal cholangeitis are prolonged over a period of from three to four weeks, the patient ultimately making a complete and permanent recovery.

The *treatment* is purely medical, being directed to the condition of the gastro-intestinal tract. Disappearance of the jaundice is rapidly followed by return of the bile-passages to normal.

Chronic catarrhal cholangeitis results from mild infection of the bile-tract, or from repeated attacks of acute infection. It is most commonly associated with cholelithiasis (see page 64), although it may occur as an independent affection.

In chronic catarrhal cholangeitis there is œdema and swelling of the mucous membrane with increased secretion of mucus. New fibrous connective tissue may be formed in the walls of the ducts, especially in those that are extrahepatic, as the result of round-cell infiltration. The walls usually are thicker than normal. In cases of partial or complete obliteration of the lumen, the ducts above the obstruction become markedly dilated. (See case history, page 77.)

The *symptoms* are essentially those of recurring or relapsing jaundice. All signs and symptoms presented are similar to those of acute catarrhal jaundice extended over a greater period of time. If the obstruction of the common duct is not complete, and if there is no ascending infection of the bile-passages, the disease will run a mild course with remissions in the intensity of the jaundice. There usually is no enlargement of the liver or spleen.

It is almost impossible to make a correct differential *diagnosis* in all cases. The conditions most frequently simulating chronic cholangeitis are calculous obstruction of the common duct, pancreatitis with obstruction of the bile-passages, or malignancy; and these often are the cause or the remote result of the biliary infection. In cases where the condition appears to be a continuation of a simple catarrhal jaundice, the diagnosis may be made, but with only a fair degree of certainty on account of the probability that the persistence of symptoms is due to a stone in the common duct.

The *prognosis* is modified by the associated lesions.

The *treatment* is the same as that in acute catarrhal jaundice, except in prolonged cases when the bile-passages should be explored and drained. Operation is indicated on account of the impossibility in many cases of excluding gall-stone obstruction. The operation of choice is a cholecystostomy (Chapter IX).

Suppurative Cholangeitis.—This rarely is dissociated from antecedent lesions of the biliary tract, the most common of which are gall-stones and tumors causing obstruction of the ducts. Any condition which interferes with the normal flow of bile reduces the

resistance of the ducts and makes them more susceptible to infection. The active ætiological factor is always bacterial life (page 7). The result of the infection generally is widespread, although in a few instances it may be limited to the ducts themselves. As a result of the obstruction below, there is a dilatation of the ducts, which usually are filled with bile-stained purulent material. The mucous membrane is congested and œdematous; the walls of the ducts are infiltrated, softened, and much thickened. On the surface of the mucous membrane may be seen points of ulceration. The liver usually is enlarged and softened, and often is the seat of abscesses which vary considerably in number and size (*suppurative hepatitis*, page 173). The abscesses usually are found near the ends of the radicles of the hepatic ducts. In some instances the outward pressure of the collections of pus found in the substance of the organ renders the surface of the liver irregular. In advanced cases the abscess or abscesses reach the surface and cause infection of the serous covering with a resulting perihepatitis, or peritonitis. Involvement of the pleura and lungs follows in some cases, with or without the development of a subphrenic abscess.

The onset often is insidious, and the *symptoms* may not even suggest the serious underlying condition. The previous history generally shows the presence of an antecedent infection either of the biliary passages (cholelithiasis, chronic cholangeitis), or of the general system, such as typhoid fever, pneumonia, etc. In those cases where there has been jaundice, serious infection of the biliary passages is announced by chills, fever, and sweats, which may be very severe. Generally there is loss of appetite, nausea and vomiting, and progressive emaciation. Jaundice may be entirely absent, although it is present in the majority of cases; but unless the condition is associated with other lesions that cause intense jaundice it may be very slight.

Pain of a dull aching character almost always is present. Sharp pain, severe in character, is present in those cases where there is an associated cholecystitis, cholelithiasis, obstruction by neoplasm, etc. The liver, spleen, and gall-bladder usually are enlarged, and there is tenderness of the liver and over the gall-bladder region.

Examination of the blood shows high leukocytosis with marked

increase in the polynuclear cells. The increase in the leukocytes often varies considerably during the course of the disease, being greater during and after the chills and fever.

Extension of the process causes involvement of the surrounding structures; pleurisy, pericholecystitis, *multiple abscesses of the liver*, etc., are among the usual complications. The symptoms of cholangeitis then are more or less hidden by the more severe symptoms of the complicating condition.

A *diagnosis* of suppurative cholangeitis may be made safely in those cases where chills, fever, sweats, jaundice, enlargement of the liver, gall-bladder and spleen, and a high polynuclear leukocytosis are found, in connection with some antecedent disease of the biliary tract. Suppurative cholangeitis may be differentiated from tropical abscess of the liver by the history of dysentery in the latter affection. In malaria the chills and fever are of more regular periodicity and blood examination should show the presence of the plasmodium and the absence of a leukocytosis.

The *prognosis* in suppurative cholangeitis is greatly modified by the treatment instituted, unless nature anticipates the surgeon and drains the bile-ducts through a fistulous opening between the gall-bladder or common duct and some viscus of the abdomen or thorax. Without drainage of the bile-ducts the prognosis is very unfavorable.

The *treatment* should be prophylactic by treating carefully and persistently any condition of the gall-bladder or biliary passages that might allow a subsequent virulent infection of the biliary tract. Gall-stones should be removed whenever the fact of their presence has been established (page 111). After the onset of suppurative cholangeitis, the treatment is medical and surgical, the former attempting to disinfect the biliary tract by the administration of hexamethylenamin and the salicylates, the latter draining the tract by means of a cholecystostomy or a choledochostomy (Chapter IX).

Immediate operation offers the best prognosis in cases of *acute obstruction of the common duct* from suppurative cholangeitis, as it does in the same condition produced by impaction of a calculus. By this means further infection of the liver is prevented, and jaundice is arrested before it becomes so profound as to cause deterioration of the blood.

Acute Obstruction of the Choledochus by Suppurative Cholangeitis and the Pressure of an Enlarged Lymph-node.

A young woman, aged 19 years, was admitted to the University Hospital, under the senior author's care, April, 1913. She had been perfectly well until six days previously, when she was striken with acute abdominal pain followed by nausea, jaundice, fever, and increased pulse-rate.

Diagnosis.—Acute obstruction of the common duct.

Operation by Dr. Deaver on day of admission. The supraduodenal portion of the choledochus was distended, and was opened. It contained a number of small stones, but the obstruction seemed to be due rather to the condition of the mucous membrane, which was much thickened, swollen, congested, and bleeding at the slightest touch. The lymph-node at the junction of the first and second portion of the choledochus was enlarged to the size of the distal phalanx of the adult thumb, and by its pressure increased the obstruction of the duct. The gall-bladder was small and contained several stones, as did the hepatic duct. The gall-bladder and ducts were emptied, and the gall-bladder and common ducts were drained. The patient recovered.

Cholecystitis. Stagnation of bile in the gall-bladder is the main predisposing cause of infection. Hence **the stagnant gall-bladder** may assume almost the importance of a clinical entity. Owing to the obstruction to the discharge of the bile from the gall-bladder its muscular tunic hypertrophies, the mucosa becomes somewhat thickened, and there is in the submucous, muscular and subserous coat a moderate lymphocytic infiltration. According to Aschoff and Bacmeister, the latter is not an evidence of infection but is due to increased absorption from stasis. The stagnant gall-bladder may or may not contain a calculus; usually a solitary stone is present, of pure cholesterin, formed of radially disposed crystals. The stagnant gall-bladder is filled with thick, viscid, non-labile bile, which is very dark olive green, sometimes nearly black in color.

The lesions found in cholecystitis vary from a very mild catarrhal involvement to the most virulent phlegmonous or gangrenous type. The various degrees of inflammation may be classified as catarrhal, suppurative, phlegmonous, and gangrenous. As regards ætiology there is very little difference in the various types, the result of the infection varying with the virulence rather than with the variety of the invading bacteria. It has been suggested by Kelly (loc. cit., page 819), however, that "the milder catarrhal

lesions are most commonly due to the typhoid bacillus and the colon bacillus, and the suppurative lesions to the pyogenic cocci."

In **acute catarrhal cholecystitis** the mucous membrane is œdematous and swollen, especially in the region of the neck of the gall-bladder; and the mucous membrane of the cystic duct is involved to a greater or less degree, with consequent narrowing of the lumen of that channel. The walls of the gall-bladder are thickened and œdematous and distention by retained material renders them tense. The thickening is due, according to Aschoff and Bacmeister, chiefly to inflammation of the subserous tissues of the gall-bladder. The contents of the gall-bladder usually consist of bile which is thick and tarry, or of serous fluid which is bile stained. This acute inflammation may subside quickly with drainage through the cystic duct, in which instance all symptoms will disappear very rapidly. In other cases the inflammatory process continues, producing first the suppurative and later the phlegmonous forms of cholecystitis. Sometimes as the acute process subsides, a chronic catarrhal inflammation takes its place, often with the formation of gall-stones; or acute exacerbations may occur which finally may result in gradual obliteration of the cystic duct and hydrops of the gall-bladder.

Hydrops of the gall-bladder (*hydrops vesicæ felleæ*) may be transitory or permanent. As pointed out by Kehr, acute infection of the gall-bladder and cystic duct of a mild character usually is accompanied by hydrops. With the subsidence of the inflammation, the cystic duct recovers its function, drainage of the gall-bladder occurs and the hydrops disappears. In these cases there is an œdema of the mucous membrane of the cystic duct, the valves of Heister formed by the convolutions of the mucous membrane being so swollen that obstruction results. With increased inflammation there may be superficial ulceration, and as a result of the cicatrical contraction of the walls of the duct, the lumen may be encroached upon to such an extent that there may be complete occlusion, with permanent hydrops.

The experimental work of Segrè proved that very marked changes occur in the walls of the gall-bladder from obstruction of the cystic duct. There is first a degeneration of the epithelium; this is followed by degeneration of the muscular layer; finally, all the layers become transformed into connective tissue. When

there is incomplete stenosis of the duct the muscular coat hypertrophies; in complete occlusion it atrophies and degenerates, so that the gall-bladder walls may become very thin, even translucent, and having the appearance of parchment.

The contents of the gall-bladder usually consist of a clear mucoid fluid, the secretion of the mucous membrane.

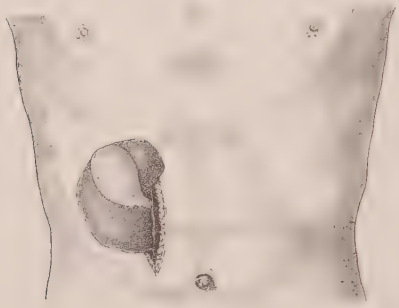

Fig. 4.—Hydrops of the Gall-bladder. (*From a patient in the German Hospital.*)

The size of the dropsical gall-bladder depends to a great extent upon the result of previous infections of this viscus and the presence or absence of pericholecystic adhesions from a prior inflammation. When the gall-bladder walls have not undergone cicatricial contraction from previous inflammatory attacks and when not bound down by surrounding adhesions, the gall-bladder may distend until it reaches the brim of the pelvis. If adherent there will be less chance of expansion; indeed it must not be forgotten that in addition to the secretion of the mucus, with which a dropsical gall-bladder is filled, there may also be a certain amount of absorption going on in the gall-bladder, and that if the processes of secretion and absorption are nearly equally balanced the gall-bladder may not become distended.

A dropsical gall-bladder is a constant menace to the patient, as it may at any time become infected, resulting in secondary empyema of the gall-bladder; while if of large size it is exposed to external injury.

Suppurative cholecystitis, or empyema of the gall-bladder, may result from a previous existing *hydrops vesicæ felleæ;* usually, however, it arises as a consequence of a virulent infection in a gall-bladder which previously had been fairly healthy, forming a more advanced stage of the so-called acute catarrhal cholecystitis already described. The condition is comparable to a suppurative appendicitis; the mucous membrane is much congested and œdematous, and may be ulcerated; there are minute miliary abscesses in the submucous, muscular and subserous coats; the

cystic duct becomes quickly occluded; and distention of the gall-bladder ensues. G. G. Ross operated upon a patient in the German Hospital, in whom the distended gall-bladder, full of pus, reached to the brim of the pelvis. The condition had been mistaken for an ovarian cyst. Such extreme degree of distention is very rare. E. M. Foote has collected five cases where the gall-bladder filled almost the entire abdominal cavity (Terrier, Tait, Gersuny, Erdmann, and Collinson). Doran collected a number of cases of comparatively moderate distention of the gall-bladder.

As the pressure increases the pus seeks an exit for itself through the cystic duct; occasionally it is discharged in this way. Usually, however, unless relieved by operation, the condition of the walls of the gall-bladder is such that perforation takes place, with or without the previous formation of pericholecystic adhesions, just as in the phlegmonous and gangrenous types of cholecystitis presently to be described. Very rarely the inflammation subsides, the micro-organisms lose their virulence and die, and the empyema is converted into a hydrops with sterile or very slightly infectious contents.

Pericholecystitis has as one of its most frequent causes suppurative cholecystitis.[1] As the purulent infiltration spreads through the walls of the gall-bladder, localized peritonitis occurs, usually as the result of perforation of miliary abscesses in the subserous coat. The gall-bladder becomes adherent to the colon, duodenum, stomach, omentum, or even to the small intestines; and occasionally *suppurative* pericholecystitis is found without any macroscopic perforation of the gall-bladder. These pericholecystic adhesions serve as a protection against the development of diffuse peritonitis; but as the acute inflammation subsides the adhesions contract, cause distortion of the gall-bladder and bile-ducts, obstruction of the pylorus or colon, and thus predispose to recurrent attacks of cholecystitis, to intestinal obstructions, etc.

Biliary Peritonitis without Perforation of the Bile-passages.—This is a condition occasionally seen, and requiring a few words of explanation. A bile-stained peritoneal effusion is found, and yet no macroscopic evidence of a perforation can be discovered any-

[1] It is desirable to distinguish between *pericholecystitis*, an active inflammatory state, and *pericholecystic adhesions* the result of previous inflammation.

where in the bile-tract. G. G. Davis called attention to the condition in 1907, reporting two cases of bile-stained peritoneal effusion without apparent lesions of the biliary tract. In one of these cases chemical examination failed to give any evidence of the presence of bile in the bile-colored peritoneal exudate, though the gauze sponges were stained characteristically yellow. Clairmont and Haberer reported a case with similar effusion, but with a stone in the common duct, yet no perforation; and Schievelbein has reported another case, in which operation was done by Ritter. In none of these cases, however, was the fluid examined for bile. Wolff has reported three more cases, and the whole subject has been reviewed by Johansson.

Clairmont and Haberer produced the condition experimentally in a few dogs out of a large number in which they caused obstruction of the common duct for another purpose: in a few days these dogs had biliary peritonitis; the abdominal wall was markedly icteric, while the skin and the rest of the muscular tissues showed only slight jaundice.

Schievelbein claims that this biliary effusion is a filtrate through the walls of a nearly normal gall-bladder. In a gall-bladder altered by disease the walls become impermeable for such filtration. He thinks the subserous position of the canals known by Luschka's name ("Luschka's Gänge") has much influence in permitting this filtration. According to Johansson, these canals exist only in about 10 per cent. of gall-bladders. It thus appears that only when the two rare factors coexist (presence of Luschka's Gänge, and acute obstruction of an almost normal gall-bladder) can biliary peritonitis occur without perforation of the bile-tract. That the effusion in some cases at least really is biliary is demonstrated by the cases of Schievelbein, and the similar case mentioned by Gibbon (Philadelphia Academy of Surgery, March, 1913), in both of which the bile-stained fluid oozed through the walls of the gall-bladder under the operator's eyes.

In **phlegmonous cholecystitis** there is a still more virulent infection. The mucous membrane becomes markedly œdematous and swollen, the submucous, muscular, and subserous coats become riddled with miliary abscesses, resulting in separation of the various coats from each other and sometimes in the separation of the entire mucous membrane as a slough. The walls of the gall-

bladder are dark in color, rigid, but friable; the contents are bloody pus, and sloughs. Usually the process is so acute that no pericholecystic adhesions are formed. The necrosis extends through the serous coat, a layer of lymph alone separating the slough from the peritoneum; when this gives way (**perforation**) the highly infectious contents are poured out into the unprotected peritoneal cavity, and diffuse peritonitis ensues.

Gangrenous cholecystitis results from the most virulent infections, or from thrombosis of the nutrient artery. The lesions are practically the same as those of the phlegmonous type except that the necrosis or gangrene involves a considerable portion of the viscus, instead of being localized to the area of perforation. Owing to the poorer blood supply at the fundus, the gangrenous process usually starts there and extends toward the cystic duct. Many so-called cases of gangrenous cholecystitis, as pointed out by Kelly (loc. cit., page 820), really should be termed ulcerative cholecystitis, a condition which usually forms a part of suppurative or phlegmonous inflammation, as already described.

Volvulus of the Gall-bladder is rare. The presence of a mesentery to the gall-bladder, resulting in the condition known as *wandering gall-bladder* (page 42), is a predisposing cause. The existence of a volvulus seldom is recognized during life; if any localizing symptoms are present they usually are misunderstood. Kübig records one case found at autopsy, and refers to three other cases reported by Mühsam, Mayer, and Fischer, in which the diagnoses made had been appendicitis, hydronephrosis, and gall-stones. All these patients were old, and three of them were women. In many cases gangrene of the gall-bladder results from interruption of the circulation. One case of torsion of the gall-bladder has been encountered at autopsy at the German Hospital:

Postmortem Record: J. McM., male, death from endocarditis. The gall-bladder was distended, its walls thickened and adherent to the liver. The cystic duct was obstructed by torsion. There was no stricture of the duct. No calculi were present.

Non-calculous cholecystitis as a pathological entity is a condition found in about 15 to 20 per cent. of all diseases of the biliary tract. In a series of 476 patients with diseases of the biliary tract operated upon by the senior author, gall-stones were absent in 18.3 per cent. Riedel found no stones in eleven of

sixty-five patients operated upon under the diagnosis of cholelithiasis.

The ætiological and pathological factors in cholecystitis have been considered at page 7.

The **symptomatology of cholecystitis** is most variable. In many instances the attack is so fleeting that the patient does not remember it and it can be recalled to his mind only after very close questioning. In other instances the onset of the inflammation is sudden and the symptoms alarming. The wide variance between these two conditions is due entirely to the virulence of the invading micro-organism and the presence or absence of an infectious disease such as typhoid fever (page 19).

All symptoms of acute cholecystitis vary considerably with the virulence of the invading micro-organism. The *pain* may be dull and aching, severe and continuous with acute exacerbations, or paroxysmal, being then identical with the so-called gall-stone colic. Sheldon collected thirty-seven cases typical of biliary colic, in which no calculi were present; and Solieri recently has reported a similar case, the pains in his patient (who had recently had typhoid fever) being caused by *hemorrhages into the gall-bladder*. A case, in which intracystic hemorrhage seems to have been responsible for attacks of biliary colic, has recently been under the care of the junior author.

Hemorrhage into Gall-bladder, Causing Biliary Colic. Cholecystostomy. Recovery.

C. D., male, thirty-seven years of age, was admitted to the Episcopal Hospital (service of Dr. C. H. Frazier), August 29, 1911, during an acute and very severe attack of abdominal colic, which was diagnosed as biliary. Had suffered pain at times after his meals, for a year or more; no hunger pain noted; no flatus in stomach; no indigestion. About one year ago passed black blood from the bowel. Ten days before admission had sudden violent epigastric and right hypochondriac pain, crampy in character. Did not vomit. Pain ceased in a few hours. For three days before admission had diarrhœa, which he attributed to drinking too freely of spring water. The day of admission at 3 P. M. while working in quarry, had a similar attack of pain so severe as to cause fainting. Four hours after admission he was rolling around his bed in agony, with hands pressed into abdomen, groaning with pain. This was colicky, came in sharp attacks, was not present all the time. Temperature 99° F. W. B. C. 12,000. Had not vomited nor even felt nauseated. The abdomen was flat, full, and rigid over gall-bladder and

appendix; no *board-like* rigidity. Extremely tender over gall-bladder and in right loin, less so over appendix; not tender elsewhere. Peristalsis audible. No urinary symptoms, no jaundice.

Diagnosis.—Biliary colic, and acute cholecystitis.

Operation, by Dr. Ashhurst, day after admission. Incision in right upper rectus; gall-bladder distended but not excessively tense; no adhesions. Pylorus, duodenum and stomach normal. Bile-ducts normal. Gall-bladder opened, and gave exit to a gush of black unclotted blood; after this was wiped up, some black tarry bile escaped, staining the gauze sponges characteristically dark olive green. Culture of bile sterile. No calculi. Gall-bladder drained by tube. Uneventful convalescence.

The initial pain usually is in the epigastrium, or it may be diffuse, gradually becoming localized in the right hypochondrium. The pain may remain localized or may be referred to the right shoulder, the right shoulder-blade, or to the right iliac fossa, in the latter instance simulating the pain of appendicitis with which condition cholecystitis may be associated.

Nausea and vomiting vary greatly. If the onset is violent there will be marked nausea and vomiting accompanied by great prostration. In mild cases there may be slight nausea and no vomiting. When the vomiting persists, the inflammation of the gall-bladder, in most instances, has extended beyond the confines of that viscus with resulting pericholecystitis or peritonitis.

Fever usually is present but varies in degree and persistency. When the infection of the gall-bladder complicates some other disease, such as typhoid fever, the temperature may be attributed to the underlying disease; if it arises during convalescence from typhoid fever the temperature may be high from the effects of the cholecystitis alone.

Muscular rigidity always is present, in some cases being of such a character that it will simulate a tumor. Careful palpation sometimes reveals the true gall-bladder tumor lying beneath the rigid muscle.

Tenderness usually is marked, and especially so in the presence of pericholecystitis. The tenderness in the beginning of the affection may be more or less diffuse, although it will almost invariably become localized over the gall-bladder.

The *gall-bladder will become enlarged*, the size of the viscus varying greatly in different cases. In some instances it is so large that it is distinctly visible. It is uniformly pear-shaped,

and moves with respiration. It may be displaced by manipulation in those cases where there are no adhesions, but always returns to its former position, thus differing from a movable kidney or a pyloric tumor. As the gall-bladder increases in size, it usually extends downward and inward toward the umbilicus; rarely it may be found in the right flank, in the right iliac fossa, or even extending to the brim of the pelvis. The subsequent history of the enlarged gall-bladder will depend upon the condition of the lumen of the cystic duct and the presence or absence of virulent infection. In the majority of cases, the cystic duct becomes patulous with the subsidence of the inflammation and the gall-bladder drains itself through the natural channels; with obstruction of the cystic duct persisting, either a hydrops or an empyema of the gall-bladder will result.

Jaundice is never seen in uncomplicated cases of cholecystitis. As long as the common and hepatic ducts remain patulous, jaundice does not occur. If the inflammation should involve either of these ducts directly or through adhesions, jaundice would ensue as soon as the lumen of the ducts became greatly lessened.

The *results of acute cholecystitis* are many; most of them are of decided clinical importance as they have a great bearing on the subsequent health and activity of the patient. In a large majority of the cases the symptoms presented disappear in from four to ten days, but it is probable that the gall-bladder seldom, if ever, returns to the condition in which it was before the infection. A chronic inflammation may ensue (page 62). A very frequent consequence of a mild, subsiding infection of the gall-bladder is the formation of gall-stones (page 9). Not infrequently the symptoms that persist after the subsidence of the inflammation are not due to the condition of the gall-bladder proper, but to adhesions formed between that viscus and adjacent organs, the result of a pericholecystitis (page 53).

When the infection is of a very severe type, or where it does not subside but rather becomes augmented, possibly by added infection, much more serious conditions of the gall-bladder ensue, *suppurative*, *phlegmonous*, or *gangrenous cholecystitis*. Of these varieties of gall-bladder disease the gangrenous is the most rare, while the suppurative type is not uncommon. In most instances the severe forms of infection occur in connection with cholelithia-

sis. Gibbon, in reporting a case of gangrenous cholecystitis, quotes Robson as speaking of gangrenous cholecystitis as extremely rare; and W. J. and C. H. Mayo as having seen but one case in 433 operations upon the gall-bladder and ducts. In a series of 328 operations on the biliary tract by the senior author, gangrene of the gall-bladder was encountered only twice. Both patients recovered.

The symptoms presented by these severe forms of cholecystitis are similar to those of acute cholecystitis, but greatly aggravated, with much more pronounced indications of involvement of the peritoneum. The pain and tenderness are more general, at times extending over the entire abdomen. Nausea and vomiting may be persistent, and the picture presented is one of infection of the general peritoneal cavity rather than of the gall-bladder. The temperature is higher, reaching 103° or 104° F. The blood count shows a decided increase of leukocytes with relative increase in the polynuclear cells. In Gibbon's case the leukocyte count was 37,600; within twenty-four hours after removal of the diseased organ, it had dropped to 12,600.

Perforation of the gall-bladder may cause a fistula between the gall-bladder and the stomach, duodenum or colon; it may open into the liver with the formation of a liver abscess; it may perforate into a mass of adhesions surrounding the gall-bladder with the formation of a local abscess. In such cases the acute symptoms are suddenly relieved. But in many cases perforation occurs into the general peritoneal cavity, and causes rapidly spreading and usually fatal peritonitis.

The **diagnosis** of cholecystitis is readily made in the majority of cases. The chain of symptoms presented in most cases, (nausea and vomiting, pain and tenderness over the gall-bladder region, rigid abdominal muscles over an enlarged and tender gall-bladder) should lead to a correct diagnosis. The mild and severe forms of cholecystitis generally may be differentiated by the intensity of the symptoms presented. It is practically impossible to differentiate between calculous and non-calculous cholecystitis in all cases, especially when the gall-stones are confined to the gall-bladder. Gall-stones, as a rule, give rise to no symptoms when in the gall-bladder, the phenomena of gall-stone colic being due to inflammation consequent upon infection. Gangrene or

perforation in acute cholecystitis cannot always be diagnosed but may be surmised on the occurence of a sudden exacerbation of the pain and increase of the general peritoneal symptoms; and perforation especially may be suspected if a gall-bladder previously palpable suddenly seems to disappear.

Acute cholecystitis must be differentiated from acute *appendicitis*, although failure to do this is not uncommon. The pain of appendicitis may be in the gall-bladder region, and the pain of the cholecystitis may be found in the right iliac fossa. Tenderness from an inflamed gall-bladder may be elicited in the appendiceal region. Both diseases may be caused by a common bacterial invasion. In patients past forty-five years of age appendicitis is comparatively rare. The pain of acute cholecystitis is more often limited to the epigastrium and the right hypochondrium, and that of acute appendicitis more often to the right iliac fossa. Referred pain is not common in appendicitis, while it often is present in cholecystitis. The initial pain in appendicitis is more general than that of cholecystitis. The presence of a tumor which moves with respiration is more suggestive of cholecystitis. We agree with Hotchkiss in attaching great differential importance to lateral compression of the lower ribs in developing pain in cholecystitis. A careful study of the onset will be a great help in clearing up the diagnosis.

In *intestinal obstruction* there is a chain of symptoms similar to that of acute cholecystitis, pain, nausea and vomiting, constipation and tympany. But in intestinal obstruction there is no rise of temperature before the onset of peritonitis; while in acute cholecystitis elevation of the temperature is common. If a gall-bladder tumor can be recognized the diagnosis is clear.

Acute pancreatitis may give rise to the same symptoms as a severe infection of the gall-bladder. In pancreatitis, however, there generally is much more constitutional disturbance and the patient appears to be more profoundly ill than in acute cholecystitis. The pain and the tenderness in pancreatitis more often are in the epigastrium. Tumor is more commonly recognized in gall-bladder infection and is located in the majority of the cases beneath the ninth costal margin, while that of the pancreas usually is behind the stomach, near the mid-line or to the left, and usually not easily palpable. Some writers claim that differentiation be-

tween these two affections is of academic interest only, as the treatment they advocate for both conditions is the same, immediate drainage of the gall-bladder. We do not agree with this opinion in all cases, however, as better results often may be obtained by proper treatment before operative measures are instituted.

Prognosis.—The prognosis in the majority of cases of acute cholecystitis is good, the inflammation subsiding and the gall-bladder draining itself through the cystic duct. It is questionable, however, whether the gall-bladder ever returns to its normal condition. We believe that a gall-bladder that has been infected will be the seat of repeated attacks of inflammation in a majority of cases. In the severe types of infection the prognosis must always be guarded, and in the gangrenous or phlegmonous type it is very grave. The sequels of the inflammation often are of much more importance than the original attack, pericholecystic adhesions frequently causing great impairment of health with marked suffering; while gall-stone formation is an exceedingly frequent sequel of infection of the biliary tract. The ultimate prognosis in these cases is greatly modified by the treatment instituted; this we believe should be operative interference, the surgical procedure being modified by the conditions found.

Treatment.—The majority of mild infections of the gall-bladder will subside under appropriate medical treatment. This implies rest in bed in the sitting position, hot or cold applications, preferably the latter, to the upper right quadrant of the abdomen, absolutely nothing by mouth and proctoclysis with normal saline solution. Before starting proctoclysis give a cleansing enema; this can be repeated each day if the patient suffers with accumulation of gas. To the enema may be added asafœtida or glycerine and turpentine. When all acute symptoms have been absent for thirty-six or forty-eight hours sodium phosphate in hot water may be given by mouth. The glycocholate and taurocholate of sodium are said to act well in aiding the return of the inflamed biliary mucosa to normal, but it is a question if they have any specific action, yet if there is no contra-indication to taking fluids, and the stomach is perfectly retentive, they can be given for if they do not do any good they will not do any harm. With the idea that hexamethylenamin is a biliary antiseptic it may be given in doses of 0.50 gramme (7 1/2 grains) three times daily.

The researches of Crowe showed that this drug was secreted not only by the liver ducts into the gall-bladder, but also by the gall-bladder itself.

The treatment outlined above is not that usually instituted by internists, but it is our opinion that this is the rational course to pursue, with various modifications, in the early stages of acute cholecystitis. We know that patients recover more rapidly and with less discomfort, *if all food is withheld for from forty-eight to seventy-two hours*, as above stated, or until there is restoration of peristalsis and the passage of gas. Repeated lavage of the stomach relieves that organ of any material regurgitated into it, and checks the nausea and vomiting. We prefer ice-cold applications to the gall-bladder region, as these allay pain and inflammation more readily than applications of heat. After recovery from the attack treatment at some of the noted Springs, such as Carlsbad, Marienbad or Saratoga, may be of value, if the case is one of non-calculous cholecystitis.

Operative procedures should be instituted in all recurrent cases of mild inflammation of the gall-bladder. A second attack of non-calculous cholecystitis is an indication that the gall-bladder is the seat of a chronic infection which is prone to flare up at any time and result in much more severe consequences. Acute cholecystitis of a severe type should be operated upon as soon as possible, except in the presence of diffuse peritonitis, when better results will be obtained by the so-called Ochsner treatment for peritonitis.

The operation of choice in non-calculous cholecystitis is simple drainage of the gall-bladder. If there is obstruction of the cystic duct due to inflammatory thickening of the mucous membrane, drainage of the gall-bladder will allow the inflammation to subside and will thus restore free communication between the gall-bladder and the common duct.

In the more severe types of non-calculous inflammation, where there is evidence that the gall-bladder will become functionless, or where the cystic duct is permanently closed by cicatricial contraction, or where there is decided enlargement of the lymph-node at the junction of the supra- and retroduodenal portions of the common duct, indicating infection of the chain of peri-pancreatic lymphatics, cholecystectomy should be performed.

Chronic cholecystitis may be the end result of acute inflam-

mation, or it may be due to an inflammation of the chronic type from the beginning. Non-calculous chronic cholecystitis is not so common as the calculous form of the disease, gall-stones being associated and sustaining the inflammation in a large majority of cases. The ætiology of this condition is practically that of the acute inflammation, "the chronicity," as stated by Kelly, "being a manifestation of lingering infection (which is common), or the consequence of very low-grade infection with almost but not quite sufficient biliary drainage."

In non-calculous chronic cholecystitis, the lesions may be confined entirely to the mucosa, with congestion, swelling, desquamation and a greater amount of mucus than normal; or there may be infiltration of the walls of the gall-bladder, with areas of erosion. In other cases the walls of the gall-bladder are distended and thin and filled with thick mucus and bile. In long-standing cases there usually is contraction of the organ; associated with these cases very frequently is a mass of adhesions surrounding and pressing upon the gall-bladder.

Chronic cholecystitis is almost always present in patients with biliary calculi which give rise to symptoms. We believe that the symptoms are caused by the inflammation and not by the gall-stones themselves. When gall-stones are present the changes in the walls of the gall-bladder usually are more marked, the inflammation invading the deeper layers of the walls and causing hyperplastic and proliferative changes. This condition is considered by Kelly as the beginning of carcinomatous degeneration.

The **symptoms** presented by chronic cholecystitis, whether calculous or non-calculous, are essentially those of cholelithiasis (page 69), and it is practically impossible to determine from symptoms alone whether or not calculi are present.

Petry suggests that it might be possible to make a differential diagnosis in many of the diseases of the liver and biliary tract by means of the olive oil breakfast, according to Volhard's technique: 200 cc. of olive oil at the temperature of the room are given to the patient in the morning, before any food has been ingested. Volhard states that the oil causes the pylorus to open and thus allow a flow of bile and pancreatic juice into the stomach. The stomach contents are syphoned out about one-half hour after the oil has been given and are then studied microscopically for calculi or fragments of stone.

The **diagnosis** between calculous and non-calculous chronic inflammation of the gall-bladder cannot be made with any degree of certainty. Repeated attacks of biliary colic followed by icterus, might lead to a correct diagnosis of cholelithiasis. In the patients of the senior author at the German Hospital, colicky pains have been noted in 70.6 per cent. of the cases of non-calculous chronic cholecystitis, while jaundice has been present in 35.3 per cent.

The *prognosis* and *treatment* are those of cholelithiasis (page 110).

REFERENCES.

Aschoff and Bacmeister: Die Cholelithiasis, Jena, 1909, S. 20.
Clairmont and Haberer: Mitth. a. d. Grenzgeb. d. Med. u. Chir., 1910, xxii, 154.
Cohen and Gibbon: Amer. J. Med. Sc., 1903, i, 210.
Crowe: Arch. Internat. de Pharmacodyn. et. de Thér., 1908, xviii, 315.
Davis: Trans. Phila. Acad. Surg., 1908, ix, 144.
Doran: Brit. Med. J. 1905, i, 1310.
Foote: Progressive Med., 1910, ii, 136.
Hotchkiss: Annals of Surgery, 1899, xxix, 435.
Johansson: Rev. de Chir., 1912, xlvi, 892.
Kehr: Gall-stone Disease; trans. by W. W. Seymour. Phila., 1901, p. 32.
Kelly: Osler's Modern Medicine, Phila., 1908, vol. v, p. 808, 819, 820, 821, 825.
Kübig: Münch. med. Woch., 1912, lix, 1998.
Petry: Wien. klin. Woch., 1911, xxiv, 928.
Riedel: Mitth. a d. Grenzgeb. d. Med. u. Chir., 1909, xx, 195.
Robson: Surg., Gyn. and Obstet., 1908, vi, 29.
Ross: cited by Deaver, Amer. Jour. Med. Sc., 1908, i, 42.
Schievelbein: Beitr. z. klin. Chir., 1910, lxxi, 570.
Segrè: Riforma Medica, 1906, xxii, 589.
Sheldon: N. Y. Med. Journ. 1905, i, 69.
Solieri: Rev. de Chir., 1911, xliii, 482.
Volhard: Münch, med. Woch., 1907, i, 403.
Wolff: Berl. klin. Woch., 1912, xlix, 2354.

Cholelithiasis.

The ætiology of gall-stone formation, the character and composition of gall-stones, etc., have been considered in the previous chapter (page 8).

Pathology.—In studying the pathology of cholelithiasis, it probably would be more correct to classify the various lesions as complications of gall-stones, rather than as changes due to the mere presence of the calculi. Gall-stones, in the absence of an added

infection, seldom cause any serious pathological changes; they may remain indefinitely in the gall-bladder, where they have been formed, without giving rise to any symptoms directly referable to the biliary tract. Kehr states that almost every tenth adult has gall-stones but that only 5 per cent. of those having biliary calculi present signs or symptoms of their presence, the remaining 95 per cent. having no knowledge of their existence. The postmortem records of the German Hospital show the presence of calculi in the gall-bladder or ducts in over 11 per cent. of the autopsies, the death in each instance having been due to diseases other than those of the biliary tract. Although these gall-bladder examinations made at autopsy are limited to 524 cases, yet the findings support the statements of Kehr so far as the prevalence of biliary calculi is concerned. The proportion of those having gall-stones and not presenting any symptoms may be as high as Kehr states; and the presence of calculi in the gall-bladder may be of no significance; yet we believe that a more careful study of such patients would elicit symptoms, in the majority of cases, due to the presence of gall-stones but incorrectly assigned to functional disturbances of the gastro-intestinal tract.

When the gall-stones are formed there are present in the gall-bladder bacteria of attenuated virulence, and a catarrhal inflammation of the mucous membrane. The gall-bladder rids itself of the catarrhal inflammation and bacteria, and the normal flow of bile becomes reestablished in a large proportion of cases, leaving the gall-stones behind as the only record of the acute process. In such instances the stones may remain in the gall-bladder indefinitely; and by themselves, unaided, will not necessarily cause any further serious trouble. The persisting lesions are so slight that they give rise to no symptoms or signs in the gall-bladder region; but there are numerous instances where disorders of digestion can be cured only by the removal of gall-stones, the presence of which has never been suspected by the patient. We believe that a recognition of these cases will greatly decrease the estimated proportion of those having biliary calculi but not presenting symptoms of their presence.

Calculi in any portion of the biliary tract invite renewed infection. Without such infection they seldom give rise to serious pathological lesions, and the gall-bladder and ducts may

remain for long periods in the condition they were in after the subsidence of the stone-forming bacterial invasion. Riedel, as quoted by Kehr, is of the opinion that a "foreign-body inflammation," independent of bacterial invasion, may arise in the gall-bladder or ducts with the same effect that would result from a "bacterial inflammation." The "foreign body inflammation," possibly, may occur; it is more probable, however, that in all such instances a bacterial invasion takes place afresh, being so rapid in its course, at times, that it does not give rise to any marked pathological lesions. This view agrees fully with the statement by Kehr that such an inflammation may burst forth and subside before the patient notices it.

Non-inflammatory movements of the concretions within the gall-bladder may be caused, to a very slight degree, by the to-and-fro motion of the bile. The lesions thus produced are of little significance, although Kehr claims that "cancer of the gall-bladder may develop from the irritation of the stones actually lying quiescent in the fundus." Stones may become dislodged from their resting place and cause some interference with the outflow of the bile from the gall-bladder. As a rule, however, they do not wander; they remain where they were formed, until dislodged by contractions of the gall-bladder incited by inflammatory irritation, the latter being the result of the new infection of the viscus or of an acute exacerbation of an old process.

The mechanical action of the gall-stones after the onset of active inflammation in the biliary tract is that of a foreign body. The concretions may remain quiescent; they may be forced into the cystic duct, or through it into the common duct, and through the common duct into the intestine. After reaching the duodenum they may be expelled with the fæces or may become so augmented by added laminations that intestinal obstruction will result.[1]

They may cause necrosis of the gall-bladder or ducts with the subsequent perforation and expulsion of the stones into the liver substance, into a mass of adhesions formed around the gall-

[1] Lediard reports a case in which there had been an indefinite history of biliary and appendicular discomfort for years. At the time of operation, eleven small stones were found in the appendix. Chemical analysis proved them to be of biliary origin. Naunyn refers to similar cases reported by Budd, Serey, Rehn, and Treves; Robson has also reported such a case. H. C. Deaver has operated on another similar case, at the Episcopal Hospital; chemical study of the calculi removed from the appendix proved them to be gall-stones.

bladder, or into one of the adjacent hollow viscera, such as the stomach, the duodenum or the colon. If they become lodged in the cystic duct, there will be a resultant closure of that channel either complete or partial, with simple hydrops or empyema of the gall-bladder, depending upon the nature and virulence of the invading organsim. At times the inflammation will subside and the cystic duct remain partially patent, so that there will be a discharge of the contents of the gall-bladder and a return to comparatively normal conditions. The presence of the stone in the duct will encourage subsequent infections, as it will always act as an irritant; it is less apt to do this if it becomes encysted by being covered by the swollen mucous membrane or by forming a pocket or diverticulum for itself. The effect of such imprisonment of the stone is to cause a deviation of the channel and consequent interference with the natural to-and-fro motion of the bile.

The stone may become lodged in the common duct and may remain there an indefinite period without giving rise to any symptoms. On the other hand, it may cause complete and temporary or partial and permanent obstruction of the duct, with damming up of the bile, changes in the gall-bladder and liver, intermittent icterus, etc.

In a series of 549 cases of cholelithiasis, operated upon by the senior author at the German Hospital, the concretions were found in the following locations:

In the gall-bladder alone in	316	or 57.5	per cent.
In the gall-bladder and cystic duct	63	or 11.4	per cent.
In the gall-bladder and common duct	60	or 10.9	per cent.
In the gall-bladder and hepatic duct	1	or 0.18	per cent.
In the gall-bladder, cystic duct and common duct	3	or 0.5	per cent.
In the gall-bladder, common duct and hepatic duct	4	or 0.7	per cent.
In the cystic duct alone	41	or 7.4	per cent.
In the cystic and common duct	1	or 0.18	per cent.
In the common duct alone	37	or 6.7	per cent.
In the gall-bladder, cystic duct, common duct, and hepatic duct	5	or 0.9	per cent.
In the common and hepatic ducts	10	or 1.8	per cent.
In adhesions surrounding the gall-bladder	4	or 0.7	per cent.
Location of stone not mentioned in	4	or 0.7	per cent.

Weeks refers to a series of ninty-nine cases in which the concretions were found in the gall-bladder alone in 84 per cent.; in

the gall-bladder and common duct in 10 per cent.; in the common duct alone in 5 per cent.

Of far greater importance than the mere presence of the stones in any case of cholelithiasis is the infection that may be added. While it is probably true that a number of those patients who possess gall-stones never present symptoms severe enough to call their attention to the biliary tract it is undoubtedly a fact that these concretions render persons so affected more liable to infection of the biliary tract than are those who are free from gall-stones. Of 476 patients with disease of the biliary tract, operated upon by the senior author, 389 or 81.7 per cent. had gall-stones. When infection is added to existing biliary calculi, the resulting pathological lesions will depend upon the virulence of the invading micro-organism and the modifying resistance of the patient. They will vary from a mild condition of catarrhal inflammation to a most rapid, wide-spreading gangrenous process. Lesions in all parts of the biliary tract and adjacent structures may be produced which will result in impairment of health if not in the death of the patient. As Kehr says: "Gall-stone disease enters upon a period of latency and can in this quiescence repose until death puts the man to sleep in the eternal rest of the grave."

Any of those lesions discussed in the previous section (Cholecystitis and Cholangeitis) may arise in cases of cholelithiasis; but the pathological processes in cholelithiasis are more or less modified by the previous inflammation of the bile-passages, and by the mechanical action of the calculi. The gross pathological lesions found in a series of 538 cases of cholelithiasis operated upon by the senior author at the German Hospital were as follows:

Lesions in 538 Cases of Cholelithiasis

	Cases.	Per Cent.
Cholecystitis, mild or absent[1]	328	60.9
Cholecystitis, acute, with catarrh, ulceration or gangrene[2]	166	30.8
Hydrops of the gall-bladder	26	4.8
Carcinoma of the gall-bladder	7	1.3
Pericholecystitis with abscess	7	1.3
Contracted gall-bladder, embedded in the liver	1	0.18
Cholecysto-gastric fistula	2	0.36
Adenomatous degeneration involving liver	1	0.18
	538	99.82

[1] In forty-seven cases there was chronic inflammation and contraction of the gall-bladder.

[2] In sixty-four of these cases pus was present in the gall-bladder (empyema).

In the vast majority of cases the attention of the patient is first called to the presence of gall-stones by a new invasion of the biliary tract by bacteria, or by an acute exacerbation of a chronic inflammation. The acute attack may be very mild and fleeting in its course and may subside so rapidly that it may soon pass entirely from the patient's memory. With each attack, however, changes occur in the gall-bladder causing that viscus to pass through the various stages of inflammation until it finally becomes contracted, with marked thickening of its walls. During the progress of these inflammatory processes, the gall-bladder may change very decidedly in shape and size. Distinct pockets or diverticula are formed in some instances; or a contracting cicatrix will cause an hour-glass formation; at other times the viscus will almost disappear, its remnant forming a tight capsule or covering for one or more calculi. These anomalies have been discussed at page 42.

In so-called "*simple cholelithiasis*," the symptoms of which are in reality due to a chronic catarrhal cholecystitis, the changes in the walls of the gall-bladder may or may not be very marked. There may be slight enlargement of the organ with some thickening of its walls. The mucous membrane usually is somewhat thickened, possibly œdematous and injected. As a rule the bile is thicker than normal, ropy, and dark in color. These conditions are not serious and usually subside so soon as the excitants of the infection, the gall-stones, have been removed. In the absence of operative interference the gall-bladder and cystic duct only in the rarest instances clear themselves of calculi; in such circumstances the bladder and duct may resume their normal condition. Even when, after the subsidence of an exacerbation, the concretions remain in the gall-bladder and cystic duct, or in the gall-bladder alone, the pathological lesions present may be such that they will not for many years give rise again to symptoms pointing to gall-bladder trouble.

This condition is well illustrated in the following case:

"Latent" Gall-stones; Acute Cholecystitis, Recurrent; Cholecystectomy. Recovery.

A. K., female, aged fifty-four, admitted to the German Hospital April 27, 1907. Married thirty-five years. Father, uncle and grandfather died of

carcinoma; one aunt living with carcinoma; mother died of hæmoptysis. Menstrual history negative. Two pregnancies. Menopause at forty-seven. Has had measles, diphtheria, varioloid, typhoid fever, and pneumonia. Typhoid attack twenty-nine years ago.

Present Illness.—First attack of sharp pain in epigastrium and over gall-bladder region in 1872, when nineteen years of age. The attack began on the sixth day of puerperium about midnight and was very severe, the pain being referred around the left costal margin to a point between the scapulæ, and being accompanied by nausea. The pain was so severe that morphia was required. Had had attacks similar to above at irregular intervals since 1872, with periods of quiescence for five or ten years. *The longest interval between these attacks was seventeen years.* The last attack began early on March 31, 1907. Pain very severe, referred around each costal margin and to each scapula.

On Admission.—Heart and lungs negative. Liver dullness extends from sixth intercostal space to two fingers' breadth below costal margin. Pear-shaped tumor can be palpated in gall-bladder region; tumor tender and moves with respiration.

Operation.—April 27, 1907. Ether anæsthesia. Incision through right rectus muscle. Stomach and pylorus normal; liver studded with whitish, elevated, circumscribed areas; gall-bladder enlarged; calculi felt in gall-bladder and in cystic duct. Gall-bladder opened and stones removed. As it was impossible to remove those in the cystic duct, the gall-bladder and cystic duct were removed.

Pathological Report.—Chronic and acute inflammation of the gall-bladder.

Patient discharged in good health, May 13, 1907.

The Cholesterin Gall-bladder.—Moynihan has given this name to certain gall-bladders whose walls seem normal on casual inspection, and the mucous lining of which feels healthy to the palpating finger. The contents are the usual dark and tarry bile of the "stagnant gall-bladder"; but on close inspection there are found imbedded in the mucosa quantities of cholesterin, as fine as sand; this infiltration of the mucosa stops abruptly at the cystic duct. Sometimes minute crystals of cholesterin may be seen glistening on the gauze with which the gall-bladder contents have come in contact; and these will give a clue to the true condition of the gall-bladder walls. It will be recalled that according to Aschoff and Bacmeister pure cholesterin stones may be formed without bacterial action; and Moynihan considers such gall-bladders as these typical examples of such manufactories. The cholesterin sand cannot be removed. The gall-bladder must be extirpated.

Pericholecystitis.—When the infection is of a more virulent type, the resulting pathological lesions will also be of more moment. The inflammation will extend through the walls of the gall-bladder and affect the visceral peritoneum, a *pericholecystitis* resulting. After reaching the peritoneum, the inflammation may extend to all of the adjacent viscera, resulting in adhesions matting together the gall-bladder, the omentum, the colon, the duodenum, the liver, the stomach, and the ileum into an unrecognizable mass. The acute inflammation may again subside but the changes wrought will be lasting. Permanent alterations in the gall-bladder will be noted, and even if the concretions within the gall-bladder remain more or less quiescent after the subsidence of the inflammation, symptoms of distressing nature will persist, due to the adhesions and the effect of their contraction.

Perforation of the gall-bladder is a rare event in cases of cholelithiasis, probably because the walls of the gall-bladder have become resistant from the long-standing infection, which from the first has been of a low grade of virulence. If, however, a virulent infection is added to a gall-bladder already containing calculi, perforation may occur precisely as in non-calculous phlegmonous cholecystitis (page 55). In the case of calculous cholecystitis, however, the long duration of a mild infection or the recurrence of attacks of subacute inflammation usually has been sufficient to produce pericholecystic adhesions, so that perforation into the unprotected peritoneal cavity is extremely rare. Thus when a perforation occurs, the contents of the gall-bladder, and not unfrequently the calculi themselves, are discharged into the surrounding adhesions, forming a **pericholecystic abscess,** or even into some adjoining hollow viscus (stomach, duodenum, colon) if the portion of the gall-bladder affected by perforation has been closely adherent to such organ, and the sloughing or ulcerative process has not been arrested spontaneously before penetrating the walls of the adherent viscus. Sometimes the gall-bladder ulcerates into the liver, and an intrahepatic abscess results. In a few instances the adhesions will be very slight, a mere "cob-web," and the stone or stones will appear to hang upon the external surface of the gall-bladder. If the perforation has taken place into a mass of adhesions, a tumesence will be formed involving any part or all of the upper right quadrant of the abdominal

cavity; the inflammation may extend upward toward the diaphragm, forming a subphrenic abscess;[1] it may burrow toward the right kidney space and simulate very closely a perinephric abscess of renal or spinal origin. Two such cases have been reported by Estes: the true origin of the condition was recognized only after exploratory incision. A rather unusual course for the pus to take in these instances has been reported by Jones: in this case the pus burrowed through a mass of adhesions into the right kidney space, forming a perinephric abscess; and this abscess opened into the pelvis of the kidney, biliary calculi being passed from the urinary bladder. Communications of the biliary passages with the pleural cavity and bronchi are considered in connection with the subject of biliary fistulæ (page 144).

A large majority of ulcerative perforations of the gall-bladder occur in those cases which have been neglected after the onset of the acute attack. When the infection is very virulent it may be possible for the succeeding steps of inflammation, ulceration, and perforation to occur so rapidly that operative treatment cannot be instituted; but usually the course of the inflammation is slow enough to allow the removal of the calculus or calculi with drainage of the gall-bladder or cholecystectomy. Early operative treatment of gall-stone cases would in the majority of instances prevent the occurrence of perforation of the gall-bladder and its numerous sequels.

CALCULI IN GALL-BLADDER; PERICHOLECYSTIC ABSCESS WITHOUT PERFORATION. CHOLECYSTOSTOMY. RECOVERY.

W. B., male, age forty-five years; admitted to the German Hospital October 10, 1904. Never had typhoid fever. Had "intermittent fever" eleven years ago. For past three years has had colicky pains in gall-bladder region with tenderness; no nausea or vomiting. Never was jaundiced; stools never clay-colored. For past two months pain has been severe, but no typical biliary colic.

Examination.—Liver extends 3 inches below ribs. Mass size of lemon palpable in gall-bladder region. Marked tenderness over mass. Œdema of abdominal wall over mass. Hæmoglobin, 64 per cent.; R. B. C., 3,700,000 W. B. C., 7,600.

Operation, by Dr. Deaver. Ether anæsthesia. Incision splitting fibres of upper right rectus muscle. Adhesions and plastic exudate between gall-

[1] This is much more often secondary to suppurative hepatitis (page 164), though Viollet has recorded a case without direct invasion of the liver.

bladder, stomach, liver, transverse colon and duodenum. Two small abscesses found, one subhepatic and one in adhesions between gall-bladder and stomach. Gall-bladder enlarged, walls thickened. Contained few calculi and pus. Adhesions broken up, abscess cavities cleaned out, concretions removed. Gall-bladder and abscess cavity drained with rubber tube and gauze, respectively. Recovery.

Calculi in Gall-bladder; Ulcerative Cholecystitis without Perforation; Subhepatic Abscess; Cholecystostomy. Recovery.

C. S., female, aged fifty-seven years; admitted to German Hospital October 19, 1905. Family history negative. Never had enteric fever. For two years had complained of indigestion, "biliousness," and epigastric distress. For seven months had severe pain near right costal margin, referred to back and groin and recurring at irregular intervals. On admission pain referred to groin, but not to back or shoulder. Vomiting present. Had not been free from jaundice for some time.

Examination.—Tumor presents beneath right costal margin, very tender on palpation. Tumor has cystic feel, and moves with respiration. Liver extends from sixth rib to umbilicus.

Operation, by Dr. Deaver. Ether anæsthesia. Incision splitting fibres of the upper right rectus. Adhesions between gall-bladder, omentum and colon, and between parietal peritoneum and liver. Gall-bladder distended and seat of ulcerative inflammation. About 200 calculi in gall-bladder. Adhesions broken up, revealing abscess beneath liver containing about 200 c.c. of bile-stained pus. Pus wiped away, calculi removed, gall-bladder drained with tube, and abscess cavity with gauze. Recovery.

Calculi in Common Duct; Perforation of Gall-bladder into Liver; Subphrenic Abscess; Abscess in Gastro-hepatic Omentum. Drainage of Gall-bladder and Common Duct. Death from Septicæmia.

D. E., male, aged forty-one years; admitted to the German Hospital November 9, 1905. Has had stomach and liver trouble at irregular intervals for past twenty years. Attack of heart trouble four years ago. Complains of dull pain below ensiform, which began eleven days ago. Pain referred to back and to right iliac fossa. Vomited once.

Examination.—Liver extends from fourth rib to costal margin. Tenderness in epigastrium and over gall-bladder. Rigidity over gall-bladder. Respirations upper thoracic and principally left sided.

Operation, by Dr. Deaver. Ether anæsthesia. Incision splitting fibres of upper right rectus. Adhesions throughout right hypochondrium. Gall-bladder enlarged but collapsed. Communication between gall-bladder and abscess under diaphragm. Abscesses in liver, and pus in gastro-hepatic

omentum. Three calculi in common duct. Pus evacuated; calculi removed from common duct by incision; gall-bladder and common duct drained by separate tubes; gauze drain to subhepatic space. Death twenty-five days after operation from speticæmia and exhaustion. No autopsy.

Perforation of the gall-bladder *into the free peritoneal cavity* usually results in a rapidly spreading peritonitis. Very seldom does it happen that the extravasated bile is sterile, or so slightly infectious as to produce no acute inflammatory reaction. Garrè, in 1904, found reports of about fifty cases of perforation of the gall-bladder or ducts in cases of cholelithiasis: in thirty-three cases the perforation occurred in the gall-bladder, in one in the hepatic duct, in five in the common duct, and in four in the cystic duct; while in the remaining cases the site of perforation was not mentioned. It is our impression that the perforation usually is at or near the neck of the gall-bladder in calculous cases, while in non-calculous cases the perforation is more apt to be near the fundus of the gall-bladder.

Cholelithiasis; Perforation of Gall-bladder into free Peritoneal Cavity; Diffuse Peritonitis. Cholecystostomy. Recovery.

M. A., female, aged sixty-three years; admitted to the German Hospital May 15, 1912. Chief complaint: great pain in center of body, back and front; it extended from the shoulder blades down as far as level of pubis.

Previous History.—Patient has had ten children and three miscarriages. Husband living and well. Patient had "indigestion" twenty years ago, and "catarrh of the bowels" thirty years ago. About this same time she had a very severe attack of "malaria." This lasted for one year, having chills every other day, but did not stay in bed. Her indigestion was so bad that she could and would eat only starch, eating a pound a day of ordinary washing starch; at times would eat only camphor. Was hungry always, but would eat very little for fear of causing indigestion. Often had attacks of "pleurisy."

Present Illness.—In December, 1911, had very loose bowels, and vomited for a week. This attack was thought to be brought on by eating cold meat. In April, 1912, she had pain in center of her trunk, front and back, for one entire day, and was sore for a week afterward. May 11 she had soreness in lower abdomen, this spread all over the trunk, and caused her to vomit until May 15. About 4 A. M., May 14 she developed a very severe pain in the lower abdomen; it came suddenly and did not yield to sedatives. The next day the abdomen was greatly distended and she was sent to the hospital.

Examination.—Abdomen greatly distended, bowels have not moved for four days. No peristalsis audible.

Operation by Dr. Ross, day of admission. Ether anæsthesia. Longitudinal incision through upper right rectus. Free fluid spurted on opening peritoneal cavity. Fluid looked like bile, and some was found in pelvis which looked like pus. About 250 gall-stones were taken out of peritoneal cavity and gall-bladder. The calculi were all sizes and shapes; the largest about the size of a hickory nut. The gall-bladder presented a perforation. It was inflamed and quite large and thickened, having adhesions in many places. The bile-ducts were patulous. Gall-bladder drained with tube, and subhepatic space drained with gauze and tube. Pelvis drained through suprapubic stab-wound. Recovery uneventful, fistula closing in six or seven weeks.

Hydrops of the Gall-bladder, already discussed at page 51, also occurs when a calculus becomes so fixed in the **cystic duct** that the lumen of the channel is occluded. The effect on the gall-bladder is the same as when this duct is occluded by cicatricial contraction, but there is much greater likelihood of patulency being restored in the former than in the latter case. In some instances the stone will cause sacculation of the duct, the channel being markedly deviated around the obstruction. When the obstruction is complete the gall-bladder becomes distended with a clear mucoid fluid, the secretion of the mucous membrane. In cases of hydrops there is seldom more than one calculus present —that causing the obstruction. This is a radial cholesterin stone; for other (secondary) calculi to form it would be necessary, according to Aschoff and Bacmeister, for bile to be present. Since both in experimental ligation of the cystic duct, and in malignant obstruction of the cystic duct (lesions presenting the same mechanical conditions as impaction of a stone) the gall-bladder rarely becomes distended (hydrops), but usually shrivels up, it is assumed by Aschoff and Bacmeister that in cases where hydrops does occur there must be some factor in addition to the mechanical closure of the duct and they suggest that perhaps there is a low-grade hæmatogenous infection present.

Calculi in the common duct cause pathological lesions that are far more serious than those produced by stones in any other situation. The condition becomes systemic rather than local, from the direct influence on the physiological activities of the hepatic ducts and their radicles, through absorption by the lymphatics along the duct; and through the effect on the gastro-intestinal tract by interference with the functions of the pan-

creas and the intestines. In exceptional cases no systemic effect is produced, the stone remaining in the duct or passing through it into the duodenum without giving rise to other than transitory symptoms, and no serious pathological lesions resulting.

The relative frequency of stones in the common duct is shown by the table at page 67. Although they were found in the common duct alone in not quite 7 per cent. of cases, yet in over 21 per cent. of all cases there were some calculi in this situation. Robson found calculi present in the common duct in over 39 per cent. of patients under his care. The more careful the search, and the more experienced the surgeon, the oftener will stones be found in the deeper ducts. As Terrier said, biliary surgery tends to become more and more "canaliculaire."

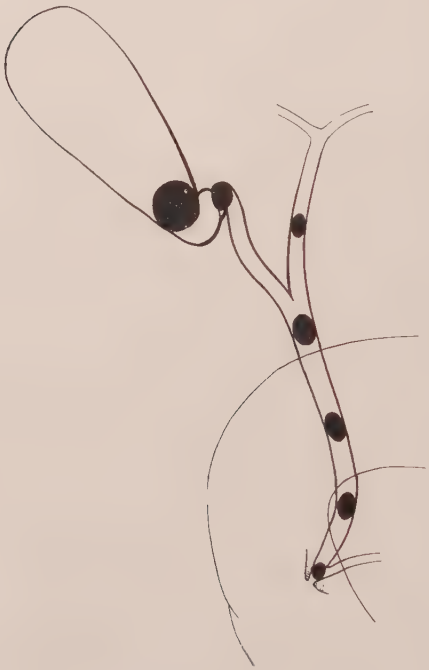

Fig. 5.—Diagram to show Various Sites of Biliary Calculi.

The position of the stones in the common duct varies from its beginning to its termination in the ampulla of Vater. Courvoisier found the stone, in an analysis of 123 cases,

At the commencement of the duct in seventeen cases.
In the middle part of the duct in nineteen cases.
Near the retroperitoneal part of the duodenum in twenty cases.
At the ampulla of Vater in forty-one cases.
Scattered along the entire common duct in twenty-six cases.

Langenbuch says that in two-thirds of the cases the stone is in the duodenal portion of the common duct. A calculus not infre-

quently projects into the duodenum through the opening of the ampulla of Vater. Calculi in this situation usually are small; indeed Moynihan claims that the further down in the duct the calculus is found, the smaller it is apt to be, the larger stones almost invariably being found in the upper portion of the duct.

As already stated, the vast majority of biliary calculi originally form in the gall-bladder, and are thence discharged into the cystic, and eventually into the common bile-duct; and when the first stone has become impacted in the common duct, all others descending from the gall-bladder will be dammed up behind it, until the complete chain may reach beyond the orifice of the cystic duct and fill the hepatic ducts also. It is of course not impossible, when a stone once has lodged in the common duct, that other calculi not derived from the gall-bladder may be formed subsequently in the ducts on the hepatic side of the obstruction. Such calculi, however, are almost invariably bilirubin calcium stones, originally of very minute size (sand), which have descended from the intrahepatic ducts, and which subsequently increase in size by lamination.

The number of stones in the common duct varies from one to an indefinite number. Though Courvoisier found a single calculus present in two-thirds of all cases of common duct lithiasis, yet at operation it is much more usual to find multiple calculi. Robson in one case recovered as many as eighty-eight stones from the common duct. The greatest number removed from the common duct by the senior author is 258. The history of this patient is of interest also in showing the size to which the common duct may be dilated.

F. X., female, aged thirty-four years. Admitted to the German Hospital, January 16, 1910. *Family history* negative. *Past history* negative. Has had five children; never has had typhoid fever. *Present illness:* for about nine years has been troubled with indigestion, having epigastric pain with a sensation of stoppage of food in this region; much eructation of gas. Since October, 1907, has been having irregular attacks of pain in right hypochondrum, occurring about once in two months. The pain at times is very severe, radiating to the back and spine, and requiring morphine for its relief. Last attack began January 10, 1910, and then for the first time patient became jaundiced. The pain is unaffected by posture or eating. Has seldom been nauseated, seldom vomited. Since onset of jaundice stools have been clay-colored. Patient was under treatment at Carlsbad for three months last year.

Physical Examination.—Well developed and nourished. Complexion markedly jaundiced, of a lemon tint. Liver dullness extends to costal margin. Mass felt under costal margin in mid-clavicular line, small in size and tender. Slight rigidity. No other mass. No abdominal distention. Hæmoglobin 82 per cent.; R. B. C. 3,410,000; W. B. C., 8,150. Coagulation-time ten minutes. Urine contains few granular and hyaline casts. Bile test strongly positive. Cammidge reaction positive. Stool: well-formed masses of fatty consistency, white, acid; occult blood negative; soaps, fatty acid crystals, fat, bacteria, small amount of vegetable material, few leukocytes and epithelial cells.

Operation.—Ether anæsthesia. Incision through upper right rectus. Adhesions between gall-bladder and omentum. Adhesions freed and ligated. Gall-bladder incised and fifty-five stones removed. Common duct found to be distended to size of small intestine and filled with bile and calculi. Bile removed with hypodermic needle. Duct incised and 258 stones, varying in size from a millet seed to a large pea, were found. Obstruction found in ampulla and stone size of hickory nut removed in pieces, with the scoop. Gall-bladder, common and hepatic ducts drained.

Patient made a quick recovery. Drainage fistula completely closed and jaundice was almost absent by time of discharge from the hospital, twenty-seven days after operation.

Complete occlusion of the common duct by a stone is comparatively rare; partial obstruction is much more common. In many instances the gall-stone remains in the duct without causing any definite symptoms of obstruction, its presence in the duct being discovered only at the time of operation undertaken for suspected stones in the gall-bladder. When obstruction takes place there occurs a damming up of bile with dilatation of the common and hepatic ducts and their radicles. Dilatation of the duct will occur gradually to such an extent that the bile may escape around the stone or stones, with consequent decrease of the jaundice. This intermittent type of jaundice was explained by Fenger by assuming that the calculus acted as a ball-valve in the common duct, floating loose so soon as a sufficient accumulation of bile had occurred behind it, and again becoming impacted as this bile escaped on the temporary relief of obstruction. But this purely mechanical explanation is invalidated by later operative experience which has shown that in many of such cases, if not in all, the stone is so firmly fixed in the duct that by no stretch of the imagination could it have been assumed to have been acting as a ball-valve.

In the patient whose history is noted above, the common duct equalled the small intestine in size; Langenbuch refers to a case recorded by Schüppell in which the diameter of the common duct was 5 cm.; and Moynihan (loc. cit., page 188) mentions a specimen in the Museum of Guy's Hospital in which the diameter of the common duct measured 6 inches. This dilatation of the common duct may be saccular, but usually is cylindrical.

Saccular cystic dilatation of the common bile-duct is comparatively rare; the condition sometimes is described as *a cyst of the common bile-duct*. Lavenson was able to find only twenty-eight cases in the literature, to which he added another. It is much more frequent in the female than the male, the proportion being about 8 to 1. In the cases collected by Lavenson the age was stated in twenty-two instances; the average was fifteen years and eight months. Two patients were under one year of age and two were between forty and fifty years of age.

The underlying *cause*, as noted above, is some form of obstruction or obliteration of the duct on the distal side of the enlargement. In the young this obstruction may be due to a congenital condition in which there is either a valve-like fold at the duodenal end of the duct or an angular insertion of the duct into the duodenum. Congenital obliteration of the common duct (page 39) might give rise to such a cystic formation, were life prolonged a sufficient length of time. In nineteen of the cases collected by Lavenson the causes of the obstruction were as follows: gall-stones in three; papilloma in one; myomyxomatous polyp in one; scirrhous pancreatitis in two; catarrhal cholangeitis in one; a simple stenosis or obliteration of the peripheral end of the lumen in six; a valve-like fold or angular insertion of the duodenal end of the lumen in five. Lavenson states that in addition to the obstruction of the lumen of the duct, a general weakness of the musculature of the duct is present. Schlössmann has collected sixteen cases of what he considers "idiopathic" cystic dilatation of the common duct.

The dilatation of the duct may vary from that of the index finger to a cyst of enormous size. Weiss reports a case in which the dilated duct contained 800 c.c. of slightly cloudy biliary fluid. It simulated an echinococcus cyst.

The *symptoms* of cystic dilatation of the common duct are

those of obstruction to the normal flow of bile. According to Schlössman the symptoms presented are merely intermittent jaundice which may continue for years; either colic-like or continuous abdominal pain which seems to vary with the jaundice; and a tumor in the right upper quadrant.

It is practically impossible to make a differential *diagnosis* in all cases. An enlarged gall-bladder or a cyst of the pancreas will present the same tumor-like mass and in the presence of jaundice nothing better than a tentative diagnosis can be made. We strongly advise against exploratory puncture in a suspected case, as was done by Schlössman, on account of the great danger of causing peritonitis. Exploratory laparotomy is a much more surgical procedure.

The *treatment* is purely surgical; but the statistics collected and analyzed by Lavenson and by Schlössman show a very high mortality. Of twenty-one cases collected by Lavenson in which operative interference had been instituted, simple puncture was done in three, with no recoveries; incision and drainage was done in fourteen cases, with one recovery; and cholecyst-enterostomy was performed in four cases with three recoveries. Judging from these findings, the last-named procedure gives the best hope for recovery.

Courvoisier's Law.—In spite of enlargement of the ducts in common duct obstruction, the gall-bladder itself seldom is enlarged; as a rule it is contracted. This is in accord with what is known as *Courvoisier's Law*[1] (loc. cit., S. 58) that in 80 per cent. of the cases of obstruction of the common duct due to stones there is contraction of the gall-bladder; while in 90 per cent. of the cases of enlargement of the gall-bladder the obstruction is due to causes other than stones.

Robson gives the following reasons for this condition:

1. All cases of cholelithiasis producing symptoms are accompanied by inflammation of the biliary passages, as shown by the almost universal presence of adhesions around the gall-bladder.

2. Gall-stones in the common duct seldom cause complete obstruction, either because they are floating in the duct or be-

[1] Often known also as the Courvoisier-Terrier Law, because the phenomenon was pointed out independently by Prof. Terrier in 1891 before Courvoisier's monograph was known outside of Germany.

cause they only partly fill it. There is, therefore, no sufficient backward pressure to cause dilatation of the gall-bladder.

3. The muscular coat of the gall-bladder contracts in efforts of expulsion when there is any obstruction in the common duct.

4. The contraction, from being at first intermittent, becomes in the long run constant, and accompanying inflammation fixes the gall-bladder, which atrophies as a result.

The second of the above reasons given by Robson is considerably invalidated by the fact that the dilatation of the common duct itself and of the hepatic ducts demonstrates the existence of

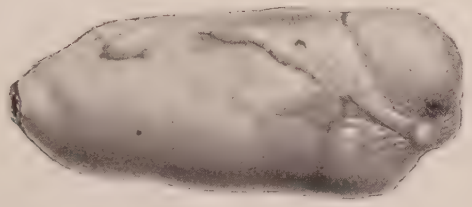

FIG. 6.—EXTREME CONTRACTION OF GALL-BLADDER, THE RESULT OF LONG-STANDING CHOLECYSTITIS. (*From a patient in the German Hospital.*) (Twice Natural Size.)

back pressure; while the fourth reason which is given as a corollary of the third, implies that the gall-bladder is in a condition of spasticity, which is far from being the case, as its muscular walls are destroyed by inflammatory infiltration long before this stage of cholelithiasis is reached. The explanation given by Courvoisier himself is sufficient to account for most cases: *that the long-standing inflammatory condition of the gall-bladder, whence in most instances the stones originated, has caused cicatricial contraction before the common duct becomes invaded.*

Violations of Courvoisier's Law.—Moynihan claims that Courvoisier's law is violated (1) where there is a stone or stricture in the cystic duct causing hydrops or empyema, together with impaction of stone in the common duct; (2) where there is a stone in the cystic duct pressing upon the common duct (see case history, page 122); (3) where there is distention of the gall-bladder by an acute inflammatory process, with obstruction of the common duct by stone; (4) where there is chronic induration of the head of the pancreas with stone in the common duct; (5) where there is ma-

lignant disease of the common duct at any part of its course, or cancer of the head of the pancreas, and a chronic sclerosing cholecystitis.

The following case history illustrates the conditions most frequently found in cases of *obstruction of the common duct by calculus.*

L. B., female, aged thirty-one. Admitted to the German Hospital, January 16, 1907. Family history negative. Married thirteen years. Four pregnancies, two miscarriages. Bowels habitually constipated. Had typhoid fever eight years ago, at which time she had marked pain in the gall-bladder region.

Present illness began in March, 1906, with severe colicky pains in the right hypochondrium, radiating to the back and right scapular region, and accompanied by vomiting. Confined to bed for one week, then up for a week and again confined to bed for eight weeks. Had constant pain in the gall-bladder region, with exacerbations of pain. Was able to be out of bed until September, 1906, during which period she did not feel well, had marked constipation, no appetite, and would perspire freely on exertion. In September was seized with excruciating pain in right hypochondrium, similar to first attack, but much more severe. Did not vomit. Had chill before onset of pain. Denies presence of jaundice in either attack. Following this attack was fairly well until two years before admission to hospital when she had attack of pain similar to the former attacks. Did not know she was jaundiced until told so. Great depression of spirits, appetite very poor. Bowels markedly constipated. Jaundice of skin and scleræ. Considerable tenderness over gall-bladder region. Lower border of liver palpable below costal margin. Abdomen soft except in the right hypochondrium where there is some rigidity. Hæmoglobin 80 per cent., W. B. C., 5900. Coagulation time, two minutes.

Operation, January 19, 1907. Ether anæsthesia. Incision through upper right rectus. Adhesions between gall-bladder, duodenum, great omentum and transverse colon. Gall-bladder thickened, contracted and empty. All ducts thickened and surrounded by adhesions. Hepatic and common ducts greatly dilated. Common duct opened below junction of cystic and hepatic ducts. Probe passed into ampulla of Vater where a calculus was found. Calculus worked upward and removed through opening in duct. Rubber drainage introduced into common and hepatic ducts. Cystic artery and cystic duct ligated, gall-bladder removed. Gauze drainage along bed of gall-bladder and into subhepatic space. Wound closed to drainage with iodine gut. Jaundice disappeared three days after operation. Recovery uneventful. Culture of gall-bladder showed pure growth of Bacillus typhosus.

Enlargement of the liver generally follows obstruction of the common duct, the smaller biliary radicles becoming much dilated; in the course of time they may even produce a sort of varicose appearance on the surface of the liver (see case history, page 70).

The back pressure eventually causes atrophy of the hepatic cells, and along with inflammatory changes due to infected bile, is responsible for the formation of new connective tissue in the substance of the liver, surrounding each biliary radicle. This is followed by *atrophy* of the parenchyma of the liver, so that there eventually is found a relatively increased proportion of connective tissue to the secreting parenchyma, and the liver becomes decreased in size. This condition closely resembles chronic cirrhosis, but Kelly distinguishes between the present condition, which he terms chronic hepatitis, and true cirrhosis.

Calculi in the common duct frequently are productive of marked *changes in the pancreas*. These are considered in Chapter VI. Extension of the common duct infection may occur by contiguity also to the *portal vein*, and rare cases are on record of portal and even splenic thrombosis, and of suppurative hepatitis due to embolism from these sources (Langenbuch, loc. cit., S. 223).

Calculi in the hepatic duct are rare. In the senior author's series of 549 cases of cholelithiasis (page 67), there was only one in which the stones were found in the hepatic duct alone; in twenty cases (3.6 per cent.) calculi in the hepatic ducts existed coincidently with stones in other locations, but with the one exception noted above in every case of hepatic duct stone there were also calculi in the common duct. In one other case not included in this series the senior author removed a single large stone from the hepatic duct. Courvoisier found that among fifty-nine cases of stones in the hepatic duct there were also stones in other locations in no less than fifty-six cases. These figures make it evident that in the vast majority of cases calculi in the hepatic duct have not orginated in that situation but have either migrated first from the gall-bladder and cystic duct into the choledochus, and have thence floated up into the hepaticus; or, what is certainly much rarer, have formed in the choledochus or in the common hepatic duct as a result of calculous or other inflammatory obstruction of the lower portion of the choledochus. Langenbuch (loc. cit., S. 216) considers it certain that while biliary sand (bilirubin calcium) may form primarily in the finer hepatic ducts and be excreted with the bile, yet that calculi worthy the name originate in the gall-bladder and reach the hepatic duct only secondarily.

The pathological changes induced by stones in the hepatic duct are not to be distinguished from those due to common duct lithiasis.

REFERENCES.

Aschoff and Bacmeister: Die Cholelithiasis, Jena, 1909, S. 50.
Courvoisier: Path. u. Chir. der Gallenwege, Leipzig, 1890.
Estes: N. Y. Med. News, 1905, lxxxvii, 1206.
Fenger: Amer. Jour. Med. Sc., 1896, i, 125, 286.
Garrè: Deutsch. med. Woch., 1904, i, 47.
Jones: Brit. Med. Jour., 1907, i, 933.
Kehr: Gall-stone Disease, trans. by W. W. Seymour, Phila, 1901, p. 25, 28.
Kelly: Osler's Modern Med., Phila., 1908, vol. v, p. 838.
Langenbuch: Chir. d. Leber u. Gallenblase. Stuttgart, 1897, Bd. ii, S. 223.
Lavenson: Proc. Path. Soc., Phila., 1909, n. s., xii, 73.
Lediard: Lancet, 1907, i, 83.
Moynihan: Annals of Surgery, 1909, ii, 1265 (Cholesterin gall-bladder).
Moynihan: Edinb. Med. J., 1906, xix, 410.
Naunyn: Treatise on Cholelithiasis. Trans. of New Sydenham Soc., London, 1896, p. 160.
Robson: Gall-Bladder and Bile Ducts, N. Y., 1904, p. 278.
Robson: Lancet, 1906, ii, 1768, Case 4 (Gallstones in appendix).
Schlössmann: Deutsch. Zeit. f. Chir., 1911, cix, 160.
Terrier: Rev. de Chir. 1892, xii, 897.
Terrier: Bull. et Mém. Soc. de Chir., Paris, 1907, xxxiii, 17.
Viollet: cited by Langenbuch, loc. supra. cit., S. 236.
Walker: Trans. Amer. Surg. Assoc., 1905, xxiii, 306.
Weeks: Trans. Amer. Surg. Assoc., 1904, xxii, 232.
Weiss: Berl. klin. Woch., 1909, xlvi, 1843.

Predisposing Causes.—*Age.*—It is impossible to determine definitely at what age biliary calculi are formed, because no one can decide how long they may, or may not, remain quiescent after their formation. Between 50 and 60 per cent. of the cases that come to operation are over forty years of age and less than 1 per cent. are under twenty. It is our belief, based upon clinical experience, that most gall-stones are formed between the ages of twenty and forty, when the system is most liable to the other infectious diseases such as typhoid fever, appendicitis, etc. In an analysis of 606 cases of cholelithiasis operated upon by the senior author at the German Hospital, the ages of the patients at the time of operation were grouped as follows:

Between 20 and 30 years	90	or	14.5 per cent.
Between 31 and 40 years	178	or	30.0 per cent.
Between 41 and 50 years	159	or	26.2 per cent.
Between 51 and 60 years	127	or	20.9 per cent.
Between 61 and 70 years	48	or	7.9 per cent.
Between 71 and 80 years	1	or	
	603		

In three cases the age was not given. This series places the age of 268, or 44 per cent. of the patients, between twenty and forty years; and 335, or 55 per cent. over forty years. When the histories of these latter cases are studied it is seen that the majority of them had had symptoms of the condition, for the relief of which they applied to the surgeon, for periods ranging from a few months to fifteen or twenty years. It is not at all unusual to note in the history that the "present illness" began five or ten years before admission to the hospital. If the time of the onset of the present illness were taken as the time of the formation of the gall-stones, it would be clearly demonstrated that cholelithiasis is a disease of early adult life and not of advanced age; and if it be remembered that even these first symptoms may not appear until years after the formation of the stone, this supposition is strengthened. MacWilliams in an analysis of ninety-seven cases of cholelithiasis gives figures which support this view:

	Cases.	Per Cent.
Between 20 and 30 years	11	11.3
Between 31 and 40 years	35	36.0
Between 41 and 50 years	22	22.7
Between 51 and 60 years	22	22.7
Between 61 and 70 years	6	6.1
Over 70	1	1.0

Forty-six, or 47.4 per cent., were under forty years of age at the time of operation. O. Hartmann found that while the average age of his male hospital patients at the time of operation was forty years, the average duration of their symptoms had been six years; while in his private practice the average age at the time of operation was thirty-seven years, and the average duration of symptoms nine years. As Moynihan points out, this carries the age at which

the gall-stones were formed back to thirty-five years in all cases. Moynihan's own patients had an average age at the time of operation of forty-five years, with an average duration of symptoms of five and one-half years.

Douglas seems to regard cholelithiasis as other than a disease of advanced age when he states that "the formation of biliary calculi is too generally regarded as incident to middle and advanced age. Lamentable errors occur from a failure to appreciate its possible existence at all periods of life."

The above-mentioned opinions are at variance with the teachings of many of the older writers. Hoppe-Seyler states that "the increasing frequency of gall-stones with advancing years is very striking. Friedr. Hoffman, Morgagni, Haller, Coe, J. P. Frank, and others have emphasized this fact, and it is established by all autopsy reports. As the latter alone furnish conclusive evidence, we are justified in believing that such a connection exists." The figures quoted by Hoppe-Seyler may be seen in the following table:

INCIDENCE OF GALL-STONES AT AUTOPSY ACCORDING TO AGE.

Period of Life	Gallstones present at Autopsy in Cases of Peters	Schröder	Rother
Under 20 years		2.4 per cent.	
20 to 30 years	0.62 per cent.	3.2 per cent.	3.0 per cent.
30 to 40 years	3.24 per cent.	11.5 per cent.	
40 to 50 years	4.44 per cent.	11.1 per cent.	6.9 per cent.
50 to 60 years	6.98 per cent.	9.9 per cent.	
60 to 70 years	9.53 per cent.		
70 to 80 years	13.02 per cent.	25.2 per cent.	19.2 per cent.
Over 80 years	16.36 per cent.		

It is probable that these investigators were greatly influenced by the incidence of gall-stones at various ages. Necropsy reports are the only ones that are reliable in determining the prevalence of gall-stones at different periods of life; but, on the other hand, they are just as unreliable in determining the part age plays as an ætiological factor in the formation of gall-stones, for the reason that biliary calculi may remain latent for years after their formation without giving rise to symptoms that will call attention to their presence. Hoppe-Seyler states that "in many, in fact the majority of the cases of concretions within the gall-

bladder or the bile-passages, all symptoms are absent, and the condition is only discovered at autopsy. Whenever the stones remain in the location where they are formed, *i.e.*, in the gall-bladder, they usually produce no symptoms." Kehr voices this same opinion in the following words: "If with Riedel we assume that in the German Empire alone two millions have gall-stones, then indeed only 100,000 feel anything of their stones, while 1, 900,000 remain exempt from pain."

As has already been pointed out, catarrhal inflammation of the gall-bladder is believed to be one of the necessary factors in gall-stone formation; and the typhoid bacillus and the colon bacillus which are the organisms found most frequently in cholelithiasis, are most active in the human subject during the period between twenty and forty years of age, when typhoid fever, appendicitis, catarrhal inflammation of the intestines, etc., are most frequent. These two bacteria were found in 37.2 per cent. of the bacteriological examinations made by Kelly in patients with cholelithiasis operated upon by the senior author at the German Hospital, while 55.4 per cent. of the cultures remained sterile (page 3).

While, perhaps, it is not susceptible of proof, it is our opinion that the great majority of gall-stones are formed during early adult life, between the ages of twenty and forty; that most of these patients present no urgent symptoms until later in life; and that the addtion of the small minority of the cases of gall-stones formed after forty years of age to those formed under forty years of age, will account for the increasing incidence of symptoms from biliary calculi with advancing years.

There is no age at which gall-stones may not be found, John Thomson even claiming, according to Still, that most of the gall-stones of early infancy are prenatal in origin, an infection of the biliary tract taking place during intrauterine life which in one instance will cause obliteration of the bile-ducts, in another the formation of stones. Still has collected twenty-three cases of cholelithiasis in children under fourteen years, including three cases of his own; fourteen of the collected cases were under ten months of age; several were in still-born infants; his own three patients were each less than nine months old.

The United States Vital Statistics Report, census of 1900, gives the following proportion of deaths due to biliary calculi.

Under 1 year of age	26 or	8.0 per thousand deaths.
Under 5 years of age	32 or	9.9 per thousand deaths.
5 to 14 years of age	11 or	3.4 per thousand deaths.
15 to 24 years of age	55 or	17.0 per thousand deaths.
25 to 34 years of age	223 or	68.7 per thousand deaths.
35 to 44 years of age	464 or	143.0 per thousand deaths.
45 to 64 years of age	1457 or	449.1 per thousand deaths.
65 and over	1002 or	308.9 per thousand deaths.

Douglas quotes Naunyn as having found, in 9000 autopsies, gall-stones in one out of every thirty under the age of thirty years, and one out of every six over sixty years of age.

Sex.—Sex has a most decided influence on gall-stone formation. The clinical experience of the senior author places the preponderance of women over men nearly as four to one. Of the 606 cases of cholelithiasis mentioned above, 461 or 76.1 per cent. were females and 145, or 23.9 per cent. were males. Schröder and Rolleston agree that the disease is more common in women than in men, although their proportions differ widely, the former giving it as five to one, the latter as four to three.

INCIDENCE OF GALL-STONES AT AUTOPSY, ACCORDING TO SEX.

Author	Male	Female
Brockbank	2.9 per cent.	7.9 per cent.
Fiedler	4.0 per cent.	15.0 per cent.
Roth	4.7 per cent.	11.7 per cent.
Rother	3.9 per cent.	9.9 per cent.
Schröder	4.4 per cent.	20.6 per cent.

The greater frequency of gall-stone disease in women is due to conditions which favor stasis of bile in this sex. Chief among these is the habit of wearing tight corsets. Rother has found corset-liver in 40 per cent. of women suffering from gall-stones. But even when tight lacing is not carried to the degree necessary to produce a corset-liver, limitation of the excursions of the diaphragm, caused by any constriction of the waist, interferes with the natural discharge of bile from the gall-bladder (see Vol. I, page 39).

Pregnancy also is important as a predisposing factor in cholelithiasis; nor does its influence cease with the emptying of the uterus. While this organ is enlarged during the child-bearing

period pressure is exerted on all the abdominal viscera, and the free evacuation of the gall-bladder is hindered by this pressure. After pregnancy terminates there often is a ptosis of the abdominal organs with twisting or kinking of the cystic duct, caused by traction, or at times, by torsion. Cholelithiasis seems to occur much more frequently in those women who have been pregnant, and especially in those who have borne many children, than in those whose uterus has never been gravid. Schröder found that ninety-nine out of 115 women, who had died during the child-bearing period and who had had cholelithiasis, had been pregnant at some time during their lives; he was able to determine that eleven of the 115 women had never been pregnant. Mayo says "90 per cent. of married women who have gall-stones have borne children, and 90 per cent. of these women identify the beginning of symptoms with some particular pregnancy."

The more frequent occurrence, in the female sex, of movable kidney, especially of the right kidney, is also regarded as a predisposing cause of gall-stone formation, since by traction on the duodenum the bile-ducts may be displaced or kinked (page 139).

Climate.—From statistics that have been gathered from numerous sources by various investigators, it has been demonstrated that cholelithiasis is much more frequently seen in some localities than others. The cause of the greater frequency in these localities is not the climate or the altitude, however, but rather the different modes of living and the greater prevalence of infectious diseases which cause catarrhal conditions of the gastro-intestinal and biliary tracts. This is demonstrated in the localities of Basle and Strasburg, which are noted for the prevalence of typhoid fever, and in which cities, according to Posselt, there are twice as many cases of cholelithiasis as in certain other European cities.

Race.—This also seems to be a predisposing cause of cholelithiasis. Our clinical experience places the Hebrew race in the foreground in relation to the frequency with which gall-stones are found among the different races. Probably this is to be attributed to their sedentary habits. The African race, on the other hand, seems to be practically immune from all gall-bladder affections.

Occupation.—*Sedentary habits* must be placed in this category,

as has been proved by the statistics of Krauss, Beadles, Gorstell, Snell and others, who have found gall-stones exceedingly common among teachers, clergymen and the insane. Krauss places among brain workers more than 50 per cent. of the 472 cases of cholelithiasis which he analyzed. This frequency among that class of workers is due to the confinement which is so closely associated with it, and not to the cerebral activity itself (page 8). The combined statistics of Beadles, Gorstell, and Snell, as quoted by Douglas, show that cholelithiasis is present in 21.22 per cent. of autopsies performed upon the insane. In these various classes, there is a tendency to stasis of the bile, a condition necessary for the formation of gall-stones. Anything, in fact, which interferes with the normal flow of bile will act as a predisposing cause of gall-stone formation. Among these factors may be mentioned gastrectasis, gastroptosis, displacement of the right kidney, constipation, marked obesity, any tumor in the region of the gall-bladder pressing upon it or the bile-ducts, etc.

This same condition is induced by *cardiac lesions* which cause stagnation of the bile, creating a catarrhal condition of the mucous membrane, by rendering the patients sedentary in their habits, etc. Rolleston shows by the report of the Manchester Royal Infirmary, analyzed by Brockbank, that gall-stones are found twice as often in those patients who have cardiac lesions prior to death as in those without such heart conditions. Among 504 autopsies showing gross cardiac lesions, calculi were present in fifty-five, or 10.9 per cent. Among 843 autopsies not showing cardiac lesions, calculi were found in forty-six, or 5.4 per cent.

The presence of any *foreign body in the biliary tract* may act as a predisposing cause of gall-stone formation, as it may become the nucleus of the subsequently formed calculus. Intestinal parasites, fruit kernels, silk threads, and other foreign bodies have been noted by Kehr as forming nuclei of biliary concretions. The researches and experiments performed by Mignot, as quoted by Moynihan, prove that foreign bodies may remain in the gall-bladder indefinitely if they are free from germ-life without causing inflammation or precipitation of the solids of the bile. There must be added an attenuated bacterial infection to cause the formation of calculi. H. Florcken reports eight cases of so-called recurrent cholelithiasis in which a silk ligature inserted at a prior

operation became the nucleus of a subsequently formed calculus. Eastman reports a case similar to one seen by Nauchè in which a steel needle formed the nucleus of a gall-stone. Eastman could not account for the presence of the needle in his case, but surmised that it had been swallowed and had passed directly from the stomach to the gall-bladder through a mass of adhesions which were present (see also page 12).

Any *septic condition*, especially in the abdominal cavity, predisposes to infections of the biliary tract, by acting as a focus from which bacteria may be carried to the bile-passages, through the systemic or the portal circulation. If the infection is attenuated when it reaches the gall-bladder, gall-stone formation may result, if the other essentials are present.

Peritoneal adhesions, from any cause, will act as a predisposing cause of gall-stone formation by favoring stasis of the bile, through distortion or kinking of the bile ducts.

Degeneration of the *thyroid gland* and the *ovaries* is a predisposing factor in gall-stone formation, according to the views of Lorand, who claims that these glands govern oxidation and obesity and that degenerative changes in them will cause atony of the biliary ducts, pathological alterations in the liver, atony of the intestine, constipation, etc., with resulting stagnation of bile.

Exciting Causes.—The exciting ætiological factor in cholelithiasis used to be assigned to heredity, to cirrhosis of the liver, to gout, to rheumatism, to faulty metabolism, to errors and indiscretions in diet, to excessive use of alcohol, to digestive disturbances, etc. Modern science has removed all of these from the domain of exciting factors of cholelithiasis and has placed them either in the category of predisposing causes, or entirely without the realm of ætiological factors, whether exciting or predisposing. There is but one exciting cause of gall-stone formation and that is an invasion of the biliary tract by attenuated bacterial infection in the presence of a stasis of bile.

A bacterial study of gall-stones shows the presence of bacteria in about 33 per cent. Thus, Funke in a bacteriological study of 102 biliary calculi obtained a growth in bouillon from thirty-one, while seventy-one cultures remained sterile. Such studies, however, are of no value in determining the action of bacteria as

the underlying factor in gall-stone formation. It has been stated by numerous investigators and has been proved experimentally both in animals and in the test-tube, that gall-stones may rid themselves of bacteria after their formation; and, on the other hand, that biliary calculi may absorb bacteria from the surrounding bile. Thus it is quite possible to obtain colon bacilli from a gall-stone whose formation was due to typhoid infection, or *vice versa;* to obtain sterile cultures from biliary concretions universally acknowledged to owe their origin to bacteria, and to obtain cultures of bacteria which had nothing to do with the formation of the particular stone from which they were grown. No reliance should be placed on the results of bacterial investigation in the case of stones that have been examined months, perhaps years, after their formation. If, however, bacteriological examinations were to be made soon after the formation of the calculi, while they were still soft, and before the gall-bladder had had an opportunity to rid itself of bacteria, micro-organisms would be found in every calculus with very few, if any, exceptions. According to Funke soft concretions invariably yield bacteria.

REFERENCES.

Beadles: See Douglas, loc. cit.

Brockbank: cited by Robson, Dis. of Gall-bladder and Bile-ducts, N. Y., 1904, p. 221.

Douglas: Surg. Dis. of the Abd., Phila., 1903, p. 343.

Eastman: J. A. M. A., 1909, i, p. 1660.

Fiedler: cited by Moynihan.

Florcken: Deut. Zeit. f. Chir., 1909, xciii, 310.

Funke: N. Y. Med. Jour., 1906, ii, 1077.

Gorstell: See Douglas, l. c.

Hartmann, O.: Zeit. f. klin. Chir., 1903, lxviii, 230.

Hoppe-Seyler: Nothnagel's Encyclopædia: Dis. of the Liver, Pancreas, etc., Phila., 1903.

Kehr: Gall-stone Disease, tr. by W. W. Seymour, Phila., 1901, p. 25.

Krauss: See Douglas, l. c.

Lorand: Arch. Gén. de Méd., 1906, i, 897; Vermont Med. Monthly, 1906, xii, 23.

Mayo: Collected Papers, 1911, Phila., 1912, p. 141.

Mc Williams: N. Y. Med. Jour., 1906, i, 1109, 1175, 1238.

Moynihan: Gall-stones and Their Surgical Treatment, 2nd Ed. Phila. and London, 1905.

Posselt: Med. Klin., 1908, ii, 1187.
Rolleston: Diseases of Liver, Gall-bladder and Bile-ducts, Phila., 1905, p. 710.
Roth: See Moynihan, l. c.
Rother: See Moynihan, l. c.
Schröder: See Moynihan, l. c.
Snell: See Douglas, l, c.
Still: Trans. Path. Soc. London, 1899, l, 151.

Symptomatology and Diagnosis.—The symptoms of cholelithiasis are presented in almost endless combinations. A study of the pathological changes due to the presence of biliary calculi with added infection shows how this is possible, and will enable the surgeon so to interpret the signs and symptoms presented that, as a rule, not only a correct diagnosis of the presence of stones may be made, but also of their exact location and of the pathological lesions which will be disclosed by direct inspection of the affected part. It is essential, therefore, for anyone who attempts to diagnose and treat diseases of the liver and the biliary tracts to be thoroughly conversant with their pathology. Without such knowledge it will be impossible to interpret correctly the signs and symptoms presented and as a consequence the patient will suffer from faulty treatment. In the majority of cases of cholelithiasis the diagnosis is easy: symptoms and signs are presented which enable one to arrive readily at definite conclusions. In the minority of cases, however, it may be impossible to make a correct pathological diagnosis; and in some cases a diagnosis can be made only after the abdomen has been opened and the affected region has been examined by sight and touch.

If the conclusions of Kehr, with which Riedel agrees, are to be accepted, 95 per cent. of the patients possessing gall-stones do not present symptoms which direct attention to their presence. We cannot agree with these findings, although the opinions of such eminent clinicians must be given great weight. We fully agree that the majority of cases do not present the symptoms commonly recognized as classical in this condition; on the other hand, we believe that symptoms are present which, until recently, have been misunderstood. Such symptoms may be elicited by studying carefully the history of the case. A carefully taken history is one of the most important requisites in every case of suspected disease of the upper right quadrant of the abdomen.

Notes of former illnesses, of "biliousness," of attacks of "indigestion," etc., should be made fully and in minute details. Inquiry regarding attacks of pain, no matter how remote, should be made, and the replies noted. The presence or absence of nausea or vomiting, of flatulence, of constipation, of fever, of chills, or of jaundice during any period in the past history should be carefully noted. Negative findings often are as valuable as are positive, in arriving at a correct diagnosis.

Many symptoms of cholelithiasis which often are classified vaguely, as stomach and liver troubles, "biliousness," "indigestion," "dyspepsia," etc., will be found in such a history, and will often lead to the diagnosis of biliary calculi before any marked symptoms have presented themselves. Riedel claims that "cramps" in the stomach are caused by gall-stones in ninety-seven out of 100 cases. When the so-called classical symptoms are present there is little, if any difficulty in making the diagnosis.

Gall-stones, unaided by infection, present practically no symptoms. All signs and symptoms presented during the course of gall-stone disease are due, we believe, to inflammation of the biliary tract resulting from infection. The symptoms vary with the severity and chronicity of the infection.

Pain is the most constant symptom of cholelithiasis and one of the most important to be studied in making a diagnosis. It is caused in all cases, we believe, by infection or the results of infection of the biliary tract and never is due to the mere presence of the gall-stones. In a series of 549 cases operated upon by the senior author at the German Hospital, pain was present in 539 or 98.2 per cent., and was absent or not obtainable in the history of only ten, or 1.8 per cent.

It is convenient to discuss the symptoms of pain in cases of cholelithiasis under the headings of *local* and *referred* pain, a plan followed by Moynihan.

Local pain, which will be considered first, is met with in two main types, the first embracing what are now generally recognized as the "gastric symptoms" of cholelithiasis, and the second being the classical "biliary colic." The "gastric symptoms" of cholelithiasis frequently are followed by symptoms of acute calculous cholecystitis without the patient ever experiencing an attack of

biliary colic. (The symptoms of acute cholecystitis have been considered at page 56.)

Gastric Symptoms.—The earliest symptom generally is pain of a character and location that is very misleading to the patient and to many medical observers. As pointed out by Richardson and as our clinical experience has shown, the patient attributes this initial pain to a bilious attack, to gastralgia, to dyspepsia, to neuritis, to gastritis, to indigestion, in fact, to anything except to gall-stones. It is described by the patient as dull, burning, gnawing, boring, grasping, etc. As a rule it is confined to the epigastrium. These "gastric symptoms" have been described as prodromal symptoms which "are said to indicate the impending formation of gall-stones" (Hoppe-Seyler). Later study has shown them to be symptoms caused by gall-stones confined to a gall-bladder in which there is very mild acute or chronic catarrhal inflammation, the latter affecting the gall-bladder alone, while the cystic duct is occluded momentarily if at all, and the to-and-fro motion of the bile is practically normal. It is recognized as gall-stone pain by the irregularity of its occurrence and by its dependence on no recognized factor. It may occur at night or during the day and be independent of the ingestion of food or of the kind of food. As Graham says "these are light attacks of distress, gas, upward pressure, coming often soon after food or at irregular times, often of sudden onset, short duration, eased by belching or perhaps slight vomiting, regurgitation, or slipping away almost unnoticed and without treatment." No particular effect is produced on the patient's constitution, and good nutrition is maintained. This initial pain is never of a colicky character.

It was pointed out in Vol. I (page 143–145) that pylorospasm, gastro-succorrhœa, etc., are not infrequently due to cholelithiasis; and we have repeatedly called attention to the fact that gastric lesions were often secondary to infections of the biliary tract. W. Soltau Fenwick has recently studied 112 consecutive cases of gastric hypersecretion, the patients having been referred to surgeons for operative relief: he found that the causative lesions were in the stomach or duodenum in seventy-five cases (67 per cent.); in the gall-bladder in fifteen (13 per cent.); and in the vermiform appendix in twenty-two (20 per cent.). He found that the cases of gastro-succorrhœa due to gall-stones occur mostly

in women past thirty years of age: there is a single large calculus; there is no history of jaundice or colic; but the patient has never been entirely well between the exacerbations of her malady. The pain occurs in the later stages of gastric digestion, in the right hypochondrium and in the right chest or back. Vomiting is not constant, but acid eructations are frequent, and the "hunger pain" is often pronounced. There is a considerable quantity of bile-stained gastric juice in the morning, in an otherwise empty stomach, the total acidity reaching forty or seventy, and free hydrochloric acid being present. After a test-meal the total acidity is much increased. When this syndrome of gastric hyperscretion is long continued, gastric and duodenal ulcers frequently develop, as a result of the prolonged contact of the mucous membrane with the acid gastric contents.

There may be slight tenderness on pressure over the gall-bladder (see page 101), but more moderate pressure in this region will relieve whatever pain is present. Some writers have noted a heavy dragging pain or sensation due to the weight of the secretions within the gall-bladder. Our clinical experience has failed to confirm such findings in any case. When this symptom occurs, some pathological lesion other than biliary concretions within the gall-bladder will be present to account for it. Pericholecystic adhesions, carcinoma or other complications will be found; and it is in such cases that referred pain occurs (page 99).

With more active inflammation of the gall-bladder, the symptoms presented are, first, those of cholecystitis (page 56); later there occur the symptoms of the complications that arise during the inflammation, or those of the sequels that follow its subsidence. As has already been pointed out a persistent chronic cholecystitis usually remains after gall-stone formation; and this chronic condition is liable to be interrupted by attacks of inflammation more or less acute in type.

Biliary Colic.—The typical "*biliary colic*" of the text-books is present in a great majority of cases that are operated upon, but it is not to be found in all of them. In the previously mentioned series of 549 cases of cholelithiasis, pain of a distinctly colicky character was noted in 462 or 82.3 per cent.; and was unrecognized by the patient at any time in ninety-seven or 17.6 per cent. This colicky pain is caused by muscular contraction

of the gall-bladder or ducts, and is not due directly to the movements of a stone either in the gall-bladder or through the ducts, although the latter may add to the severity of the colic. It is well known that in exceptional cases biliary calculi are recovered from the fæces of patients who have never suffered from "biliary colic"; and that attacks of biliary colic are very seldom followed by the passage of a calculus; while every surgeon of experience has operated on patients who have never passed any stones in spite of numerous attacks of colic, and yet in whom no gall-stones were to be found at the time of operation.

Biliary colic, in other words, is the equivalent of renal colic, appendicular colic, and intestinal colic: in all varieties the pain is not primarily caused by the passage of a foreign body, but by disordered and violent peristaltic action of the diseased organ, which has been excited in the effort to overcome an obstacle to its evacuation. This obstacle is rarely a foreign body in the nature of a calculus; almost invariably it is due to some other form of obstruction, either stenosis of the lumen from acute inflammatory œdema, to kinks produced by displacement of the organ affected, or to viscosity of its secretion. There is distention of the gall-bladder or ducts with bile and mucus, and an attempt to force a passageway through the duct. If an obstruction in the cystic duct is relieved by a stone returning to the body of the bladder or by the subsidence of the inflammatory obstruction at the mouth of the duct, the pain disappears at once, the gall-bladder emptying itself through the duct. In the mildest cases the pain may be fleeting; of such short duration, in fact, that the patient will soon forget that he has experienced it and can recall it only after the most thorough questioning. This acute, initial attack of mild colic causes no pain, as a rule, in the region of the gall-bladder—the pain is in the mid-epigastric region. In other cases, however, the initial attack is severe, sudden, overwhelming. A man, believing himself to be in the enjoyment of perfect health, except for slight gastric symptoms which have never seriously annoyed him, may suddenly have a dreadful cramp in his upper abdomen; he bends forward, pressing his hands or the back of a chair into his belly; breaks out in a cold sweat; becomes deathly pale and feels faint; is nauseated; and sometimes his distress is relieved by vomiting. At other times he

will writhe around his bed, or even on the floor, in utmost agony. Hot applications to the epigastrium or hypochondrium may have scarcely any effect on the pain, and even morphine hypodermically does not always bring relief as soon as could be wished. Death sometimes occurs during the paroxysm (Langenbuch). The fever which follows the sweat may rise to 104° F., or higher.

When the obstruction is not relieved the pain will be more lasting, remaining for hours or even days, not so intense as at first, but still severe. The pain now passes to the gall-bladder region, with pain referred to the back and shoulder. This pain is significant because it usually indicates that there are taking place, in the neck of the gall-bladder or in the cystic duct, changes which will result in permanent damage to these structures. The obstruction may be relieved by a stone passing through the cystic duct, or by the subsidence of the acute inflammation of the duct, but the lesions will be permanent and the parts cannot again return to a normal condition. Simple hydrops, empyema, gangrene, or perforation may result.

With complete obstruction of the cystic duct resulting in simple hydrops, colicky pain usually disappears; with incomplete obstruction the colic continues indefinitely. In the latter class of cases, there often is a referred pain in the right iliac fossa, at times paroxysmal and sharp, resembling in many respects the pain found in connection with an impacted ureteral calculus.

When the inflammation involves the common duct, or when a stone in its passage has reached this location, the colicky pain will be felt in the right hypochondrium, usually associated with referred pain in the right shoulder. Subsidence of the inflammation will be followed by cessation of pain, even if a gall-stone remains in the duct.

Yet even during the intervals between attacks or in the cases of patients who have had no distinct attacks of biliary colic, there is seldom a feeling of perfect comfort in the gall-bladder region. The symptoms of indigestion, of gastric flatulence, etc., after meals, will persist, perhaps becoming more pronounced; the clothes will be loosened around the waist; and moderate pressure or support with the hand to the right hypochondrium will be attempted to relieve the dragging sensation usually significant of pericholecystic adhesions, and chronic cholelithiasis. Langenbuch,

and later Moynihan, have called particular attention to a sensation of chilliness, during the evening, as particularly suggestive of mild infection of the gall-bladder.

Referred Pain.—As pointed out in Vol. I (page 25) the various positions to which pain caused by affections of the liver and biliary track may be referred can readily be explained by a knowledge of the nerve supply of the organs involved. The liver and biliary tract are supplied by three nervous systems, the *cranial*, the *spinal*, and the *sympathetic*. These connections have recently been well summarized by Millard.

The tenth *cranial*, or pneumogastric nerve, distributes sensory fibres to the œsophagus, the stomach, the lungs, and special fibres to the heart, liver, spleen, pancreas, kidneys, suprarenal bodies and the intestinal blood-vessels. The *spinal* system is represented by the phrenic nerve which is derived mainly from the fourth cervical nerve, a branch of which, the supraacromial, is distributed to the integument of the point of the shoulder. The phrenic distributes fibres to the diaphragm and falciform ligament of the liver; it unites with filaments of the cœliac plexus to form the diaphragmatic plexus which is joined by filaments from the diaphragmatic ganglion. From this plexus fibres are distributed to the coronary ligaments and peritoneum of the liver and to the right suprarenal. The *sympathetic* nerve supply is derived from the cœliac plexus, which is joined by branches from both semilunar ganglia and from the right pneumogastric. From it arise the coronary, hepatic, and splenic plexuses. The hepatic plexus inosculates with fibres from the left pneumogastric and enters the liver. Filaments are distributed to the right suprarenal; other filaments follow the branches of the hepatic artery.

On account of this general intercommunication of the nerve supply of the liver, biliary apparatus and other regions of the body, pain caused by lesions of these organs may be referred to various positions. It may be purely epigastric; may occur in the right or left hypochondrium; in either kidney region; in the diaphragmatic area, in the cardiac region, or in the left lung; in the back; beneath either shoulder blade; at the tip of the shoulder; or throughout the abdomen.

In the earliest stages of cholelithiasis, where the gastric symptoms predominate, the pain rarely is referred. Sometimes a dull

aching, a feeling of discomfort, is experienced to the right of the spinal column. When the disease has lasted for some time, and pericholecystic adhesions have developed, implicating the pylorus, the pain may be referred to the left subscapular region (Vol. I, page 3).

In attacks of biliary colic the pain is very constantly referred, either to the right shoulder, to the right chest, or more rarely to the right groin. In colic from involvement of the common bile-duct the entire lower right thorax may be painful, from swelling of the liver.

The character, location, and duration of pain are important diagnostic factors both in the diagnosis of infections of the biliary tract and in the differential diagnosis of all other lesions in the upper portion of the abdomen. The peculiarities of the pain should be carefully considered whenever present; and if absent this fact should always be noted.

Pain in "gastric symptom cases" of cholelithiasis is dull, burning, gnawing, boring, or grasping. It is confined to the epigastrium or possibly referred to the back. It is independent of the ingestion of food or of the kind of food. It may occur at night or during the day.

The pain of more severe infection of the biliary tract is colicky in character, very severe, of sudden onset, usually of short duration, and of sudden cessation. It is felt in the epigastrium or the right hypochondrium and may be referred to the breast, back, right costal arch, right shoulder, shoulder blades, etc. The attacks are independent of the ingestion of food and usually of the kind of food; they may occur at night on an empty stomach. The intervals between attacks may vary from hours or days to years.

The pain in *ulcer of the stomach* usually is of a stabbing character, located in the epigastrium, being referred at times to the left hypochondrium or left scapular region. It is rarely present when the stomach is empty, but follows the ingestion of food either immediately or within half an hour, and often increases in severity until the stomach has been emptied by vomiting or by the expulsion of stomach contents into the duodenum. The pain is seldom absent for any long periods of time.

The pain in *ulcer of the duodenum* may run in periods of at-

tacks, the periods varying in time from a few days to several months. During the periods of attacks, the pains will appear daily or several times a day. They are burning and gnawing in character, and radiate to the region of the stomach and duodenum. They bear a regular relation to the patient's meals: they are at their height from two to six hours after the ingestion of food, and are relieved or entirely dissipated by food or drink or by removal of the contents of the stomach either by vomiting or by irrigation. Between these periods of attacks the patient considers himself in perfect health; whereas in cholelithiasis a certain amount of gastric distress is constantly present.

The pain in *cancer of the stomach* is more or less continuous, of a dull, depressing character. It is generally increased by the ingestion of food.

Tenderness obtained by pressure or palpation is closely allied to pain in all cases of cholelithiasis. Some writers lay much greater stress on certain points of tenderness or pain elicited by pressure, than on any other symptom. This tenderness is claimed by some to indicate, absolutely, by its presence or absence, the presence or absence of gall-stones. Robson and Cammidge state that "another characteristic symptom of great diagnostic value is the existence of a tender spot an inch above the umbilicus, and in a line between it and the right costal margin. This tender spot is quite as constant as the McBurney point in appendicitis, although in some cases it may be a little higher than that mentioned, but in the same line." This is frequently spoken of as "Robson's point."

This characteristic point of tenderness may also be elicited in the following manner, usually called the Murphy method. With the patient sitting up and leaning forward, the examiner stands back of the patient with one hand hooked under each costal margin at the ninth costal cartilage. Deep inspiration forces the liver and gall-bladder downward while the finger-tips press inward and upward. In this way pressure on the kidney is avoided, while tenderness from the stomach or duodeum is much more superficial. Pain is experienced by the patient as pressure is exerted on the deep-lying gall-bladder and the inspiration is checked suddenly by the pain, the patient being unable to take a full inspiration. While we believe that this tenderness is very significant of the

presence of biliary calculi, it is scarcely warrantable to go as far as Bishop, who says that "if the result of such examination is negative, the possibility of biliary calculi is eliminated." Monsarrat claims that this tenderness is the one persistent clinical sign upon which a diagnosis may be made. This tender point is also elicited by "thumb pressure" under the ninth costal cartilage, after the manner practised by Moynihan (loc. cit., page 113).

Fig. 7.—Palpation of the Gall-bladder by "Thumb Pressure" under the ninth Costal Cartilage.

The left hand is placed over the lower right thorax as the surgeon sits or kneels at the right side of the patient's bed, and the thumb of this hand presses firmly but gently over the region of the gall-bladder just below the costal border. Or the surgeon may employ the method which, according to Langenbuch (loc. cit., S. 252) was originally recommended by Rheinstein (1891). The left hand is placed under the patient so as to steady the lower right thorax; then by bimanual palpation, using the right hand over the upper abdomen, the two hands are gently but firmly approximated, and as the right kidney and the liver are pushed for-

ward, the tenderness due to the diseased gall-bladder becomes evident at the end of deep inspiration.

No undue force should be used in palpating the gall-bladder region, for rupture of a diseased gall-bladder has been recorded (Langenbuch) as due to such manœuvres.

There also is very constantly a tender spot in Boas's area, on the right side behind, on the level of the twelfth dorsal vertebra, two or three finger-breadths from the spine, and this may be present even when no tenderness can be discovered in front.

Fig. 8.—Bimanual Palpation of the Gall-bladder.

While these tender spots are of significance when present, their absence does not preclude the presence of gall-stones. Our clinical experience has been unable to confirm the constancy of these diagnostic signs as observed by others. They have been absent or not obtainable in a number of cases, especially in some "gastric symptom cases" where the gall-stones and the inflammation have been confined to the inside of the gall-bladder. Where there is or has been pericholecystic inflammation, tenderness is much more constant. In such cases slight rigidity of the overlying muscles is present. while in cases of cholelcystitis, and other

acute phases of gall-bladder disease, both tenderness and rigidity are pronounced.

Enlargement of the Gall-bladder.—The gall-bladder is enlarged in the majority of cases of cholelithiasis, but the enlargement may not be sufficient to cause the presence of a palpable tumor, on account of the position of the gall-bladder in its fossa on the under surface of the right lobe of the liver. The normal gall-bladder cannot be felt through the abdominal wall; when the gall-bladder is palpable it must be distended sufficiently to force its fundus beyond the border of the liver. It is possible for the gall-bladder to be very tense without there being a palpable tumor, since the gall-bladder wall may be thickened and sclerotic from long-standing disease, and be incapable of further distention; thus it is not unusual to find a small contracted gall-bladder full of pus (empyema), and very tense. In a series of 111 cases of cholelithiasis operated upon by the senior author, the gall-bladder was distended in seventy-four, or 66.6 per cent.; it was normal in size in seven, or 6.3 per cent.; and was contracted in thirty, or 27 per cent.

The position of the gall-bladder varies with the size and position of the liver; in normal conditions, the neck of the gall-bladder is about on a level with the ninth costal cartilage. In cases of ptosis of the liver or enlargement of that organ, the neck of the gall-bladder may be as low as the umbilicus or even below that point. If the gall-bladder is enlarged the fundus usually moves downward and forward, although it is not rare to find the tumor extending toward the median line, nor is it unusual to find at operation that the gall-bladder, though enlarged, is hidden behind the liver and therefore was not palpable through the abdominal walls. In some instances it has extended to the brim of the pelvis, and in this position has been mistaken for an ovarian tumor (page 53).

When the distention of the gall-bladder is sufficient to form a palpable tumor, the latter will be felt as a rounded, smooth pear-shaped mass, readily movable laterally in the absence of adhesions. In the presence of pericholecystic adhesions, the tumor may not be recognizable as the gall-bladder, the characteristic shape and smoothness being absent. Under these conditions a mass will be palpable, more or less fixed. The mass may move

with respiration or be fixed to the anterior abdominal wall. When the gall-bladder is free there is a distinct movement with respiration, the dome of the tumor passing under the palpating hand with each respiratory movement.

In the more severe infections it is often impossible to determine the presence of a tumor, even though it may be very evident at operation, on account of the rigidity of the overlying muscles. There must be more or less laxity of the abdominal muscles in the right upper quadrant, if the gall-bladder is to be palpated.

The enlarged gall-bladder, unless held in place by adhesions, may be moved or pushed from its normal location, but will return immediately when relieved.

A tumor of the gall-bladder without jaundice usually indicates closure of the cystic duct, generally by a calculus.

A tumor of the gall-bladder with jaundice, is indicative of pressure from without, in about 90 per cent. of such cases. Jaundice due to calculous obstruction usually is accompanied by a contracted gall-bladder (see page 80). Enlargement of the gall-bladder with jaundice usually is significant of carcinoma of the head of the pancreas, of the duodenum, or of the common duct; or of benign disease of the pancreas.

Fever.—An increase of temperature at time of operation for cholelithiasis is noted in about one-third of the cases. In a series of 386 cases of cholelithiasis operated upon by the senior author, fever was absent in 264, or 68.4 per cent.; and was present in 122 or 31.6 per cent. The rise in temperature is due to an infectious process, and naturally is absent except during the activity of the infection. With the subsidence of the acute infection, the fever disappears. So long as the infection is confined to the gall-bladder there rarely is any marked rise in temperature, since the gall-bladder is nearly devoid of lymph-nodes and very little absorption occurs from it. But when the infection spreads either to the surrounding peritoneum or to the bile-ducts, then marked temperature changes occur. The temperature in cases of cholangeitis, biliary hepatitis, etc., may resemble that of malaria or septicæmia. In such cases the sudden onset of fever, its rapid rise to a height of 101° F., or more, and its quick fall again to the normal, are quite characteristic, the temperature

forming what Moynihan has well named a "steeple" chart. There is entire absence of fever between the excerbations.

Vomiting.—Vomiting usually is present during the attacks of biliary colic, and usually is absent between the attacks. At times it begins with the termination of the attack of colic; first the ingested food and later bilious matter being rejected. Blood is very rarely present. Yet hæmatemesis is occasionally seen. The vomiting often seems to relieve the pain. In rare instances the vomiting may be persistent, even alarming.

Vomiting in *ulcer of the stomach* is almost as constant as is pain in that affection. Vomiting usually brings absolute relief. The vomitus is sour, often contains an excessive amount of liquids, and is streaked with blood in over 25 per cent. of the cases.

Vomiting in *ulcer of the duodenum* usually increases in severity with the progress of the disease, at times becoming alarming. It usually occurs about two to four hours after the ingestion of food, at the time the pain and gas are greatest. The vomitus is "acid," "acrid," "bitter-burning," rather small in amount but very irritating. It frequently contains blood.

Vomiting in *cancer of the stomach* is irregular. Some relief is generally afforded. The vomitus is foul-smelling and consists of partly digested food and contains altered blood in about two-thirds of the cases.

Crepitation.—In a few instances a strong crepitation may be elicited by pressure on a gall-bladder containing calculi. LeBlanc reports such a case, in which auscultation also elicited a sound exactly similar to the crepitus of a fractured bone. Such cases are rare.

Tetany has been referred to in Vol. I (page 166) as frequently of gastro-intestinal origin. It is occasionally observed on the subsidence of a severe attack of biliary colic.

Localization of Gall-stones.—In most cases of cholelithiasis it is possible to ascertain with a fair degree of accuracy whether the calculi still remain in the gall-bladder or whether they have escaped into the bile-ducts, and if so whether the common duct is involved. As calculi may be found in several ducts as well as in the gall-bladder at the same time, the clinical picture may be somewhat confusing; but a distinction between stones still in the gall-bladder, those in the cystic duct, and those in the common

duct, usually can be made by attention to the history of the case and a careful physical examination.

The symptoms and signs of **stones in the gall-bladder** alone ("simple cholelithiasis" as it is called) are those of chronic catarrhal cholecystitis. There may be an occasional exacerbation resulting in acute cholecystitis, or in gall-stone colic; and it is chiefly on the *recurrence of symptoms* that the diagnosis of gall-stones is based. A single attack of cholecystitis, even with severe colic, is not enough to warrant a diagnosis of stone.

Stone in the Cystic Duct.—The frequency of this localization of gall-stones is shown in tabular form at page 67. Including cases where calculi are found elsewhere in the bile-tract as well as in the cystic duct, this portion of the tract is involved in about 20 per cent. of cases. The symptoms and signs vary with the size of the stone, the acuteness of the inflammation, and the completeness of the obstruction. As soon as a stone impinges upon the mouth of the duct or enters its lumen, typical gall-stone colic results from the attempt to rid the duct of the obstruction. Paroxysms of pain recur until the stone either passes through the duct, returns to the gall-bladder, or becomes lodged permanently in the duct. In the latter case obstruction is not necessarily complete, since usually a portion of the lumen remains through which bile can pass. Should the obstruction become complete, however, the pain will disappear as further attempts at dislodgment gradually cease. The pain of obstruction of the neck of the gall-bladder is referred to the right shoulder-blade in 70 per cent., to the left scapular region in 10 per cent., and to the sternum, præcordial region, and right subclavicular region in 20 per cent. of cases, according to McBurney.

Jaundice is not present in uncomplicated cases of calculus in the cystic duct. The presence of jaundice implies obstruction of the common or hepatic duct.

The presence or absence of *enlargement of the gall-bladder* depends upon the character and degree of inflammation in the organ before the stone enters the duct, and upon the completeness of the obstruction. When there has been previous inflammation, the gall-bladder walls usually are thickened and contracted; or the entire organ may be held fast by surrounding adhesions. In these cases no enlargement of the gall-bladder will occur. In

the majority of instances, however, a dilatable gall-bladder is present, enlargement occurs and the gall-bladder can be palpated. If obstruction of the duct is complete, no bile can enter, and the gall-bladder becomes distended with its secretion, forming *hydrops vesicæ felleæ*, the symptoms of which were considered at page 57. Should acute infection occur in the gall-bladder, *empyema* develops. The symptoms of this condition were discussed at page 58.

The *diagnosis* of obstruction of the cystic duct depends upon a correct interpretation of the symptoms enumerated above, and on the recognition of enlargement of the gall-bladder. When the gall-bladder is small or contracted or bound down by adhesions it is nearly impossible to make a diagnosis of obstruction of the cystic duct. Moreover, enlargement of the gall-bladder may be caused by any condition which causes obstruction to the flow of bile, or even by the presence of a large number of calculi within the gall-bladder.

Pressure on other structures exerted by a stone in the cystic duct may give rise to symptoms that will make the diagnosis more difficult. Thus cholangeitis and, at times, obstructive jaundice may result from pressure on the common duct; thrombosis and ascites may result from pressure on the portal vein; symptoms of gastric dilatation may result from pressure on the duodenum. But these conditions are comparatively rare, and the history of the case should aid in clearing up the diagnosis, especially when enlargement of the gall-bladder is present.

The *prognosis* in obstruction of the cystic duct is good, unless the contents of the gall-bladder become infected so virulently that acute empyema, perforation or gangrene results. The prognosis under such conditions is very grave. In uncomplicated cases the operative mortality is no higher than that of cholecystectomy in general.

Stone in the Common Duct.—The frequency of stone in the common duct was discussed at page 67. Including cases where calculi are found elsewhere as well as in the common duct, this is involved in over 21 per cent. of the cases. The more experienced a surgeon becomes, the less apt is he to let a stone in the common duct remain undetected at operation.

The *symptoms* presented are pain of a colicky nature, followed

in most instances by jaundice which is intermittent and which varies greatly in intensity even when present. The attacks of pain often are associated with intermittent feverish attacks, and with chills; and in most instances such attacks are followed by an increase in intensity of the jaundice. The chain of symptoms presented is similar to that of "Charcot's Intermittent Fever," a type of cholangeitis associated with obstruction of the common duct. The symptoms of cholangeitis have been described at page 47.

There is no enlargement of the gall-bladder in uncomplicated cases of obstruction of the common duct by calculus. There are few exceptions to this rule; they have been discussed at page 80.

The *diagnosis* of calculous obstruction of the common duct depends largely upon the history of the case. If the patient is seen during an attack of acute cholangeitis it may be possible to make a diagnosis even in the absence of an accurate history. Obstruction of the common duct by calculus may be differentiated from that due to carcinoma of the duct by the train of symptoms usually noted in the latter affection. Chronic progressive jaundice, without fever, with decoloration of the stools, absence of sharp pain, moderate enlargement of the liver, a distended gall-bladder, and continuous emaciation, are characteristics of obstruction from cancer of the common duct (page 123). Obstruction of the common duct from enlargement of the head of the pancreas, as a result either of inflammation or of a new growth, is considered at page 356.

The *prognosis* of calculous obstruction of the common duct is very much graver that where the cystic duct alone is obstructed. The danger is in large part due to the more serious lesions present, but in no small degree to the more extensive operative interference required to assure removal of all calculi. The mortality of choledochotomy varies from 8 to 30 per cent., in various statistics.

REFERENCES.

Bishop: Brit. Med. Jour., 1907, i, 676.
Fenwick, W. Soltau: Lancet, 1910, i, 706.

Graham: Collected Papers by the Staff of St. Mary's Hospital, Mayo Clinic, 1910, Phila., 1911, p. 41.
Hoppe-Seyler: Nothnagel's Encyclopædia, Diseases of the Liver, Pancreas, etc., Phila., 1903, p. 554.
Langenbuch: Chir. der Leber u. Gallenblase. Stuttgart, 1897, vol. ii, S. 239, 240.
LeBlanc: Med. Courier, Phila., 1906, xi, 96.
McBurney: Clinique, 1907, xxviii, 472.
Millard: Illinois Med. Jour., 1907, xi, 392.
Monsarrat: Practitioner, 1908, lxxx, 779.
Moynihan: Gall-stones and their Surgical Treatment, Phila., 1905, p. 148.
Murphy: N. Y. Med. News, 1903, i, 825.
Richardson: Bost. Med. and Surg. Jour., 1906. clv, 329.
Riedel: Path d. Gallensteinleidens, Jena, 1903.
Robson and Cammidge: Gall-stones, Their Complications and Treatment, London, 1909, p. 81.

Treatment of Cholelithiasis.

After the discussion of the pathology of this affection, into which we have gone at some length, it scarcely seems necessary to insist further on the fact that cholelithiasis is a surgical disease, and that operative treatment is requisite for its cure. Kocher's oft-quoted epigram that gall-stones belong primarily not to the surgeon but to the patient, is true enough; but the modest statement of fact which he appends should not be overlooked. It is to the effect that it is the patient's privilege, if he so elects, to spend an invalid's life sojourning from time to time at the various resorts such as Carlsbad, Marienbad, Saratoga, etc., thus cautiously endeavoring to keep his calculi quietly at rest in his gall-bladder; or even to endure with fortitude the occasional agony induced when a calculus leaves the gall-bladder and attempts to force its passage into the bowel. But this life is only for the well-to-do classes; as Kehr says, at Carlsbad the poor disappear absolutely; "the patients come to Carlsbad usually in the period of latency; the minority still have colics or inflammation of the gall-bladder. Now begins the regular living, the beneficial, pain-assuaging, laxative action of the Carlsbad springs, the delightful influence of the Sprudel baths with their peat poultices to the liver and region of the gall-bladder. The beautiful surroundings" he proceeds "entice the cure-guest into the noble forest, he climbs

the mountains, which in stillness leave nothing to wish for, and he forgets the worry of his business and the pain of his disease. The cuisine permitted by the cure removes the sins of his club life at home, of the many strawberry and peach punches; briefly, the tissue changes are powerfully stimulated, and whoever is not very sick must in a very short time indeed feel himself well." But while such a life is a patient's privilege if he can afford it, there are very many who cannot, and it is the duty of the attending physician to inform his patients that modern surgery offers a rapid and a lasting cure at a very trifling risk, provided operation is undertaken before complications have arisen. The best time for a Carlsbad "cure" is after operation.

In uncomplicated cases the mortality of operation is less than 5 per cent., and in the hands of those who have much experience in this work it has been reduced as low as 2 or even 1 per cent. When complications develop, the mortality rises rapidly, as will be noted on subsequent pages. Not only is the migration of calculi from the gall-bladder into the duct a factor of very serious moment in increasing the death-rate from cholelithiasis, but even when the stones remain in the gall-bladder the occurrence of acute cholecystitis or its possible sequels, such as pericholecystic adhesions, perforation or gangrene, may put the patient's life in jeopardy at any time. The safest course, by far, for the patient to pursue is to have his gall-stones removed as soon as they begin to produce noticeable symptoms. At this time the simple operation of drainage of the gall-bladder, with removal of the calculi, will effect a cure. Later not only cholecystectomy may be necessary, but incision and drainage of the common duct or even of the hepatic duct may be required. These operations, and others yet more complicated, have a much higher mortality, and often gravely tax the technical skill as well as the judgment of the surgeon. We believe there is no more difficult surgery than that of the bile-ducts.

The comparative mortality of operations for these various conditions may be seen in the following series of cases, published by the senior author in 1908. They comprise the operations performed in cases of cholelithiasis at the German Hospital between Jaunary 1903 and April 1907.

OPERATIONS FOR CHOLELITHIASIS (1903–1907).

Lesions.	Operations.	Deaths.	Mortality.
Simple cholelithiasis	45	2	4.4 per cent.
Calculi in ducts, but without acute inflammation	65	10	15.4 per cent.
Acute cholecystitis	54	10	18.5 per cent.
Hydrops vesicæ felleæ	12	3	25.0 per cent.
Pericholecystic abscess	6	2	33.0 per cent.
	182	27	14.3 per cent.

Kehr reported (1899), a mortality of 1.5 per cent. among his cases of cholecystostomy; 3.7 per cent. in his cholecystectomies; and of 10.2 per cent. in his choledochotomies. In extremely complicated operations, his mortality was 45 per cent.

Mayo reports (1911) an operative mortality of less than 2 per cent. when the stones were confined to the gall-bladder and cystic duct; and of 8 per cent. when the common duct was involved, varying from 3 per cent. in quiescent cases to 25 per cent. in cases of complete obstruction of the choledochus.

A series of 329 operations for cholelithiasis and its complications, by the senior author, during the years 1910, 1911, and 1912, is summarized in the following table:

OPERATIONS FOR CHOLELITHIASIS (1910–1912).

Lesions.	Operations.	Deaths.	Mortality.
Cholelithiasis without acute infection	173	9	4.6 per cent.
Acute cholecystitis	15	2	13.3 per cent.
Empyema	29	4	13.8 per cent.
Hydrops	12	2	16.6 per cent.
Pericholecystic abscess	6	1	16.6 per cent.
Gangrene or perforation	3	0	
Pancreatic lymphangeitis	24	1	4.2 per cent.
Chronic pancreatitis	67	3	4.5 per cent.
	329	22	6.6 per cent.

The cases classed as "cholelithiasis without acute infection" include all quiescent cases of choledochus and cysticus and hepaticus calculi, as well as those of simple cholelithiasis, where the stones were confined to the gall-bladder. For similar classes of cases the mortality in the earlier series of operations was nearly 11 per cent., while in the present series it has been reduced well below 5 per cent.

Recurrence of gall-stones after operation is exceedingly rare. Although, as noted at page 90, calculi occasionally have formed around a silk suture or other foreign material left in the gall-bladder at operation, usually "recurrence" implies not a new formation of stones,but that some calculi were overlooked at the time of operation. If operation is undertaken at the propitious time, that is, before any calculi have left the gall-bladder, and before pericholecystic adhesions have developed as the result of attacks of cholecystitis, then the complete removal of the calculi is not difficult, and no stones should be overlooked. This is a great argument in favor of early operation. Maurice Richardson reported a case in which, after dislodging a stone from the common duct, it escaped into the hepatic duct; and he found himself absolutely impotent to find it again: "there was the patient with his common duct wide open, and somewhere in the depths of the liver was the offending stone, ready to be drifted down into the same impaction as before." Fortunately such a catastrophe seldom occurs, even to surgeons of less skill and experience than Richardson.

Robson calls attention to the fact that in the very large experience of Mayo, Kehr, and himself, it had been stated by each separately that the recurrence of gall-stones after cholecystostomy was an extremely rare event. In 1911 W. J. Mayo stated that among 4000 operations performed upon the biliary tract by himself and C. H. Mayo only three cases were observed in which stones had reformed in the gall-bladder.

But while this freedom from recurrence is the rule, a candid statement of fact must be made that very ocasionally true recurrences of gall-stones (not cases of overlooked stones) do occur. We have encountered a few such cases ourselves. The following history showing that not very many months after the senior author had removed 100 calculi from a patient's gall-bladder another surgeon removed over 200 stones from it, is incontrovertible proof of recurrence. Two hundred calculi could not have been overlooked at the first operation.

Miss——, nurse, operated at the German Hospital in 1897 for calculous cholecystitis, removal of 100 gall-stones. Remained perfectly well for one year when she had a recurrence of cholecystitis and subsequent operation with removal of 200 stones.

The thing which demands elucidation is not why recurrences occur, but why they do not occur more often. True, these patients when operated upon have passed the age during which infectious diseases such as typhoid fever, entero-colitis, etc., are prone to occur. When they are relieved of their calculi and the existing infection of the bile-tract is cured by drainage it may be argued that no renewed infection of the biliary tract is likely to develop, because these predisposing causes of biliary infection will not again arise. Yet such renewed infections occasionally do occur. They are extremely rare, however, and would be rarer still we believe were postoperative drainage of the gall-bladder maintained for a longer time than often is the case.

But though early operation is the best treatment, in cases of *simple cholelithiasis* it may be very difficult to persuade the patient to submit to operation. If he has had no acute attacks, either of cholecystitis or gall-stone colic, and if he suffers merely from symptoms of "indigestion," he may be quite satisfied to stay as he is; and a surgeon must feel very sure of his ground before attempting to convince such a patient against his will. Of course one must bear in mind the slight but seemingly unavoidable mortality which attends any large series of operations. An occasional death will occur from pneumonia, following the anæsthetic. But as Kehr says, "*the slight dangers of early operation stand in no sort of a relation with the great dangers of the disease itself*. . . . . Even the latent cholelithiasis we should always regard with suspicious eyes, for the quiet work of gall-stones is often the most destructive." Carcinoma may develop from stones which cause no particular distress; and perforations into hollow viscera (internal biliary fistulæ, page 144) often occur without producing any acute symptoms. "The danger of carcinoma alone," writes Mayo, "is five times as great as is the mortality following operations for the relief of simple gall-stone disease." "*In malignancy and insidiousness*" concludes Kehr, "*no disease of man compares with cholelithiasis.*"

Yet operation is not to be insisted upon indiscriminately in every case. The *contra-indications to operation* may be summarized as serious organic lesions of the heart, lungs, or kidneys; extreme age; anæmia and slow coagulability of the blood. Such patients as these must lead an invalid's life, and may hope by care-

ful dieting and attention to hygiene to prevent the development of acute complications which will render operation imperative at all hazards.

What the type of operation shall be in these interval cases is a subject that has been discussed in recent years a little too strenuously. Certain surgeons contend that in every case the gall-bladder should be removed. They regard it as the hotbed of infection, and claim that its retention favors reformation of calculi. They dwell upon the supposed functional uselessness of the gall-bladder, and upon its pathological importance. They magnify the frequency of persistent fistulæ, or reformation of calculi and recurrent cholecystitis. But it is well known that calculi have developed in a dilated common duct after cholecystectomy (Mayo has observed five such cases); and the danger of fistula persisting after cholecystostomy is not great if no obstruction of the ducts remains.

It is our firm belief that the gall-bladder has important functions to perform, of which the chief are to equalize the pressure of bile in the ducts and to secrete mucus which shall dilute the bile. That it acts as a reservoir for the storage of bile has been disputed recently, but it has seemed to us that it must perform some function of the kind during long intervals between meals, as at night; and it has even been suggested by one of us that perhaps the reason that persons with gall-bladder affections sleep better after a late supper than might be expected of persons in their condition, is that the process of digestion keeps the gall-bladder more or less empty of bile, and that thus the presence of the lesser evil procures less disturbed sleep.

A further argument in favor of retention of the gall-bladder unless functionally useless is that, should a subsequent operation be required, the gall-bladder is, as stated by Hartmann, not only the thread of Adriadne which guides us through a labyrinth of adhesions to the position of the bile-ducts, but may become a very important feature in the restoration of intestinal drainage of bile, by means of cholecystenterostomy, in cases where the common bile-duct is permanently obstructed. The argument used by the advocates of indiscriminate cholecystectomy in this connection is not valid. They assert that if the gall-bladder had been removed at the first operation no secondary operation for recurrence of symptoms

would be required. Such has not been our own experience, and we agree with Richardson that cholecystectomy often is a difficult and dangerous operation—more difficult and dangerous in fact than a simple choledochotomy, were the latter operation done in cases free from pathological adhesions and in patients not gravely ill will cholæmia. The adhesions which form after some cholecystectomies cause more trouble than the original disease.

We believe cholecystectomy scarcely ever should be performed in cases of simple cholelithiasis. When, however, acute calculous cholecystitis (page 120) is present cholecystectomy usually is preferable to cholecystostomy; but in the quiet cases in which no acute inflammatory attack ever has occurred, simple drainage of the gall-bladder is sufficient. When, however, as the result of repeated attacks of acute cholecystitis, or from the long duration of the disease in a latent stage, the gall-bladder is much contracted upon its contained calculi, removal of these and drainage of the gall-bladder even if prolonged seldom will restore to it a sufficient degree of functional activity. In such cases we now prefer to do cholecystectomy at once; but we recognize it as a more serious procedure, and if the patient's condition is unfavorable, and in the case of very fat patients, where the operation is one of unusual difficulty, we still practise the simpler operation of cholecystostomy. Should further trouble occur it may be necessary to remove the gall-bladder then; but at all events we hold it is better for the patient to submit to two operations and live to tell the tale than to perish from the first. It is in such cases as these that cholecystectomy is the difficult and dangerous operation to which Richardson refers; but desperate diseases require desperate remedies, and when disabling symptoms persist the gall-bladder must be removed.

Cholecystectomy we see, then, is not indicated in cases of simple cholelithiasis with no gross pathological changes in the gall-bladder; in cases which present evidences of past attacks of acute cholecystitis, and in cases with pericholecystic adhesions removal of the gall-bladder is indicated when operation is done in the interval, unless the constitutional condition of the patient forbids. It is indicated, as we shall point out further on (1) in most cases of *acute calculous cholecystitis*, as well as (2) in *hydrops with oblit-*

eration of the cystic duct, (3) *chronic empyema*, (4) *calcareous degeneration*, (5) the *cholesterin gall-bladder of Moynihan*, described by MacCarty as the strawberry gall-bladder, (6) *gangrene of the gall-bladder*, (7) *carcinoma*, and (8) in *most cases of perforation*.

Treatment of Biliary Colic.—In many cases the pain is not so severe but that it will be relieved by local hot applications, rest in the recumbent position, and abstinence from all food. Nausea may be relieved by inducing vomiting by drinking a couple of glasses of hot water. If retching persists, lavage of the stomach may be necessary. Should the pain be great, there is no reason why morphine should not be administered hypodermically; but before this is done the surgeon should be very sure of his diagnosis. If there is a perforation of the stomach or duodenum, instead of an attack of biliary colic, immediate operation will be more effective in allaying pain than many hypodermic injections.

When the attack of biliary colic subsides, the matter of operation should be put before the patient, and he should be urged to submit to having his gall-bladder drained as the surest means of preventing a return of his colic.

Treatment of Stone in the Cystic Duct.—The operation of choice is cholecystostomy in all instances where there is a probability that the lumen of the duct will be restored after removal of the obstruction. The impacted calculus should be pushed back into the gall-bladder, whence it is removed with scoop or forceps. If the stone cannot be dislodged easily it is better to remove the gall-bladder and cystic duct than to attempt direct removal of the stone after incision of the cystic duct (*cysticotomy*). In these cases the stone almost always will be found to have caused ulceration, which will render very probable the subsequent occurrence of stricture. If the gall-bladder and cystic duct are not removed, perforation at the site of impaction in the cystic duct may occur even when the duct has been carefully sutured after the cysticotomy, and in spite of free drainage of bile from the gall-bladder. Where the duct appears to be permanently occluded, as in cases of hydrops and chronic empyema, or if the walls of the organ are much diseased, then also the gall-bladder and cystic duct should be removed.

Solitary Calculus in Gall-bladder, Obstructing Cystic Duct; Empyema; Phlegmonous Cholecystitis; Cholangeitis; Hepatitis. Cholecystectomy, Drainage of Choledochus. Recovery.

M. G., male, age thirty years; admitted to the German Hospital, June 16, 1903. Has suffered from indigestion and constipation for years. For past three years has had pain in right hypochondrium, generally dull, but at times sharp, referred to back. No jaundice. Has vomited.

Examination.—Mass felt in right hypochondrium extending from margin of ribs to umbilicus and continuous with liver dullness. Moves with respiration. Tenderness is marked over mass as is rigidity over upper right rectus. No heart murmur. W. B. C., 19,400.

Operation by Dr. Deaver. Ether anæsthesia. Pericholecystic adhesions with plastic exudate. Liver enlarged and congested. Gall-bladder distended and contained about 150 c.c. of greenish-yellow pus. Its walls were soft and mucosa gangrenous. One gall-stone of acorn size was embedded in neck of gall-bladder. Calculus with gall-bladder and cystic duct removed. Common duct drained. Recovered.

Treatment of Stone in the Common Duct.—It has long been considered unwise to operate when there is acute obstruction of the common duct; it is the opinion of the majority of surgeons in this country at least, that it is better to tide the patient over the attack under medical treatment than to subject him to the danger of an operation when so acutely ill. In Mayo's hands the mortality of operation in such cases was nearly 25 per cent. In all these cases of obstructive jaundice from impaction of stone there are times when some bile filters through and can be detected in the fæces by appropriate tests. If the patient has been carried succesfully through the stage of complete obstruction, he should be subjected to operation just so soon as the stage of incomplete obstruction is reached. Delay then certainly is more perilous than operation, though even in such cases, where acute cholangeitis persists, but where complete obstruction is not present, the mortality is about 10 per cent.

Treatment during the persistence of acute obstruction is the same as that advised for biliary colic: nothing by mouth; lavage for nausea and vomiting; local applications (hot-water bag or poultice) for pain; morphine for unendurable pain (which is rare); and proctoclysis of saline solution until the stomach becomes retentive and normal peristalsis is restored. Operation during complete obstruction may be forced upon the surgeon sometimes

by signs of perforation; but few such patients will be rescued. Operation should not be done during the existence of spreading peritonitis; it should be postponed until the process has become localized.

But though, as we have said, the majority of surgeons still hold to this teaching, we have been forced by our own experience to the conclusion that in the long run *immediate operation during an attack of acute obstruction of the common duct is attended by less danger than is delay*. If operation is delayed the patient runs the risks of cholangeitis, cholæmia, with the gravest form of sepsis; not to mention perforation of the common duct or the formation of almost inoperable adhesions; or the indefinite persistence of chronic jaundice with its dangerous hemorrhagic tendencies.

Our experience with immediate operation is so far too limited for us to be willing to erect this as a rule of practice, but while many times there has been cause to regret not operating during the stage of acute obstruction, never yet has there been cause to regret prompt relief of the obstruction by operation.

The operation of choice consists in choledochotomy, removal of the stones (which involves thorough exploration of the common and hepatic as well as the cystic duct), and drainage of the common duct and of the gall-bladder by separate tubes. Occasionally the gall-bladder has to be removed.

Choledochotomy for the removal of a calculus was first done by Kummel in 1890. It was studied at length by Terrier in 1892; he distinguished between the operation done for this purpose (*"choledochotomie proprement dite"*) and that done for drainage in cases of cholangeitis (*"choledochostomie"*). Parkes in 1885 had proposed the operation. Langenbuch in 1884 had suggested removal of a calculus impacted in the retroduodenal portion of the choledochus by an approach through the transverse mesocolon near the hepatic flexure of the colon. Vautrin, in 1896, reported three cases in which he had employed **mobilization of the duodenum** to facilitate **retroduodenal choledochotomy;** the descending duodenum was loosened from the posterior abdominal wall and turned to the patient's left. The idea of this manœuvre appears to have originated with Terrier. Haasler introduced the suggestion into Germany in 1898, and it was finally appropriated and systematized by Kocher in 1903, in connection with his

operation of gastroduodenostomy. **Transduodenal choledochotomy** to remove a stone near the ampulla of Vater, proposed in 1884 by Langenbuch, was first adopted by McBurney in 1891. Kocher in 1894 performed a somewhat similar operation, which he named transduodenal choledochostomy, as he sutured his choledochus incision to the incision in the posterior duodenal wall. Bibliographical references to all these early operations were collected with great care by Terrier in 1892.

Hepatotomy, for removal of calculi embedded in the substance of the liver, was employed in 1887 by Knowsley Thornton.

The technique of exposure of the various parts of the common duct and removal of calculi by choledochotomy is described in Chapter IX. If possible the calculus should be pushed into the most accessible portion of the common duct, which is then incised directly on the calculus. Sometimes, as urged by Kuhn, it is possible to work the calculus back into the gall-bladder if the cystic duct is dilated, and thus the necessity for opening the common duct may be obviated. When the common duct has been opened it is our custom, and we believe it of much importance, always to make sure that the duct is patent throughout by passing a sound through it into the duodenum, as insisted upon so strongly by Terrier.[1] Until this can be done the surgeon should not be satisfied that he has removed every cause of obstruction. Since adopting this practice we are quite sure that exceedingly few calculi in the common duct have been overlooked.

Drainage in these cases should be prolonged. From four to six weeks at the least should elapse before the biliary fistula is allowed to close; but it often is difficult and sometimes impossible to keep it open so long. It is too early closure of the fistula that is chiefly responsible for recurrence of cholecystitis and cholangeitis after operation.

Treatment of Acute Calculous Cholecystitis.—As intimated already, we have been forced to the conclusion that cholecystectomy is preferable to cholecystostomy in most cases of this nature. Though in cases of cholelithiasis where there never has been an attack of acute cholecystitis, cure without fear of recurrence may be secured in the vast majority of cases by cholecystostomy,

[1] Terrier pointed out that this "catheterism" of the biliary passages was first adopted in 1743, by J. L. Petit, in a case of external biliary fistula.

this is not true of the gall-bladder which is acutely inflamed. If prompt operation (within forty-eight hours of the commencement of the attack) is undertaken in cases of acute cholecystitis, the gall-bladder and ducts are free from adhesions, and cholecystectomy by the typical technique is an easy operation. Subperitoneal enucleation may be accomplished, after division of the cystic duct and ligation of the cystic artery; and the peritoneal flaps left may be sutured together, effectually covering in the bed of the gall-bladder on the under surface of the liver. This complete peritonization of denuded surfaces prevents the development of adhesions, which are almost unavoidable when removal of the gall-bladder is delayed until after the patient has had several attacks of acute cholecystitis. In such cases pericholecystic adhesions already exist, and though the gall-bladder may require removal because no longer functionally useful, the operation is much more difficult and the mortality is much higher than when cholecystectomy is done early in the first attack of cholecystitis. The benign character of the operation under the latter circumstances has been demonstrated by Leriche and Cotte.

It is in these cases of acute calculous cholecystitis that we find recurrence of symptoms, if not indeed new formation of calculi, after the operation of simple drainage; and we repeat once more that whereas in cases of simple cholelithiasis cholecystostomy is the operation of choice, in cases of acute calculous cholecystitis cholecystectomy is to be preferred.

The treatment of the other acute complications of cholelithiasis, such as *perforation*, *gangrene*, *pericholecystic abscess*, etc., is the same as when no calculi are present (page 62).

REFERENCES.

Deaver: Amer. Jour. Med. Sc., 1908, i, 37; 536.
Haasler: Verhandl. d. Deutsch. Ges. f. Chir., 1898, xxvii, i, 126.
Kehr: Gallstone Disease, Translated by W. W. Seymour, Phila., 1901, pp. 107, 112, 135.
Kehr: Trans. Congr. Amer. Phys. and Surg., 1903, vi, 212.
Kocher: Zentr. f. Chir., 1903, xxx, 33 (Mobilization of duodenum).
Kocher: Textbook of Operative Surgery, translated by Harold J. Stiles, 3d English Edition, 1911, p. 532 (footnote).

Kuhn: Münch. med. Woch., 1901, xlviii, 92.
Kummel: Deutsch. med. Woch., 1890, xvi, 239.
Langenbuch: Berl. Klin. Woch., 1882, xix, 725.
Langenbuch: Berl. Klin. Woch., 1884, xxi, 809; 826; 829 (footnote).
Leriche: Rev. de Chir., 1906, xxxiv, 110 (Mobilization of duodenum).
Leriche and Cotte: Revue de Chir., 1912, xlvi, 869.
Mayo: Collected Papers, 1911, Phila., 1912, p. 146.
Mayo: Collected Papers, 1905–1909, Phila., 1911, p. 223.
Moynihan: Brit. Med. Jour., 1913, i, 8.
Parkes: Amer. J. Med. Sc., 1885, ii, 102; Trans. Amer. Surg. Assoc., 1885, iii, 335.
Robson: Brit. Med. Jour., 1907, ii, 1117.
Terrier and Dally: Rev. de Chir., 1891, xi, 648; 1892, xii, 136, ("Catheterism des Voies Biliaires").
Terrier: Rev. de Chir., 1892, xii, 897, ("De la Choledochotomie proprement dite").
Terrier: Congrès Franç. de Chir., 1892, vi, 121, 146 (Bibliography).
Thornton: Brit. Med. J., 1887, ii, 1148 (Hepatotomy for gall-stones).
Vautrin: Rev. de Chir., 1896, xvi, 446.

Obstruction of the Biliary Passages

Obstruction of the biliary passages is of very common occurrence. The causes may be classified in three groups: (1) Obstruction from within the ducts, which is exemplified almost solely by the lodgment of biliary calculi, although rare cases of obstruction from intestinal parasites have been recorded. (2) Obstruction from changes in the walls of the ducts, which are due chiefly to strictures and neoplasms, both rather unusual causes of biliary obstruction. (3) Obstruction from pressure of neighboring structures: among these may be mentioned especially pericholecystic adhesions, pancreatic lymphangeitis and chronic pancreatitis, and carcinoma of the head of the pancreas. Other rarer causes of biliary obstruction must also be classified here—such as pyloric tumors, enlarged lymph-nodes, or kinks of the ducts caused by movable kidney. A large calculus in the cystic duct may by its pressure cause obstruction of the hepatic and common ducts as seen in the following case:

Common Duct Obstruction by Pressure of Calculus in Cystic Duct; Cysticotomy; Drainage of Cystic Duct. Recovery.

C. G., female, aged sixty-five years; admitted to the German Hospital, May 7, 1904. Father died of "liver trouble." One brother died of cirrhosis of liver. Present illness "began in childhood." Pain and colic,

jaundice, clay-colored stools. Last severe attack was two weeks ago. Pain and distress worse at night. Patient deeply jaundiced.

Examination.—Mass in gall-bladder region, which moves with respiration. Marked tenderness over mass. Systolic heart murmur. Hæmoglobin, 68 per cent., R. B. C., 3,520,000; W. B. C., 10,650. Coagulation time seven minutes. Fæces contained fat, bile and muscle-fibre.

Operation by Dr. Deaver. Ether anæsthesia. Incision splitting fibres of upper right rectus. Adhesions between gall-bladder, duodenum, omentum and liver. Liver large and cirrhotic. Gall-bladder small and atrophic. One calculus in cystic duct pressing on hepatic and common ducts. No concretions in gall-bladder or in common duct. Cystic duct incised and stone removed. Drainage of cystic duct. Recovery. At the present time this case probably would be treated by cholecystectomy.

Biliary obstruction from impaction of calculi has been considered already: the pathology of the condition was discussed at page 75, and its clinical aspect at page 118.

Strictures of the Bile-ducts.—*Congenital* obstruction of the bile-passages is discussed at page 39. *Acquired* narrowing and occlusion of the bile-passages varies in frequency with the duct under consideration. Stricture of the cystic duct is not uncommon; that of the common duct is rare; while stricture of the hepatic duct is almost unknown.

The **cystic duct** is the least unusual site for a stricture. This is so not only because of its tortuosity, but because it is the commonest site for the lodgment of a calculus; and it is the cicatrization of an ulcer, the result of the passage of a calculus, which is the usual cause of the stricture. Cordier suggests as a contributing factor also stagnation of bile in the gall-bladder, there being no constant pressure of bile to keep the duct clear, as is the case in the hepatic and common ducts.

The main *symptoms* presented are those of hydrops of the gall-bladder (page 57), or, if the contents of the gall-bladder become infected, of suppurative cholecystitis or empyema of the gall-bladder (page 58); and the *treatment* is that suitable for these conditions.

Stricture of the **common duct** generally is due to injury by the passage or attempted passage of a gall-stone or is the result of a previous operation. It is possible that a strictrue may occur as a complication or sequel either of typhoid fever or syphilis.

The *symptoms* of stricture of the common duct are those

usually seen in cases of constant obstruction of the duct (page 222), notably jaundice which is constant and increasing, acholic stools, marked indigestion with nausea, and emaciation. In the absence of infection above the stricture there should be no fever. The gall-bladder may be distended and palpable.

Strictures of the **hepatic duct** are very rare: theoretically the lesion should cause the same symptoms as those due to constant obstruction of the common duct, with the exception that the gall-bladder will not be dilated. Langenbuch refers to seven cases found at autopsy.

A *differential diagnosis* between stricture of the common duct and other forms of chronic continuous obstruction of the common duct cannot be made with any degree of certainty; nor can a distinction be drawn between obstruction of the common duct and that of the hepatic, except by the absence of a distended gall-bladder in the latter condition. But as in most cases of cholelithiasis the gall-bladder is contracted, little reliance can be placed on this sign.

The *treatment of strictures of the bile-duct* is by operation, and this should not be delayed, as every day aggravates the patient's condition. Two indications are to be met: to reestablish a channel for the excretion of the bile, and to drain, temporarily, the infected gall-bladder and hepatic ducts. It is the opinion of Mathieu, to whose excellent monograph on strictures of the biliary ducts every student of this subject must refer, that the latter indication can be attained in most cases by the former means, namely by allowing drainage into the intestine, by direct treatment of the stricture or even by cholecystenterostomy; but should there be any evidence, even the slightest, of active cholangeitis, the surgeon will have to establish independent drainage either by cholecystostomy, or by drainage of the choledochus, or the hepaticus, or even by hepatostomy (page 129).

When there is stricture of the cystic duct, with resulting hydrops or empyema, or an atrophied condition of the gall-bladder, cholecystectomy is the proper operation, combined in many cases with drainage of the common duct. When the obstruction involves the hepaticocystic juncture, cholecystectomy must be supplemented by resection of the obstructed portions of the common and hepatic ducts (Fig. 9).

When the stricture is in the common duct or in the hepatic duct, the technique of the operation for the re-establishment of the course of the bile comprises: (1) Treatment of the obstruction, when accessible, including resection or incision with plastic opera-

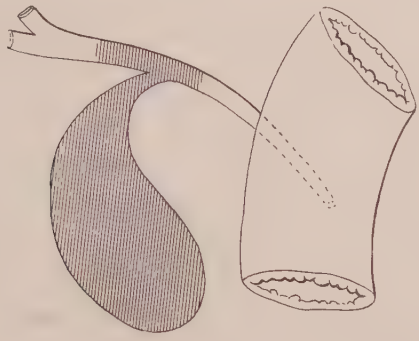

Fig. 9.—Diagram to Show Parts which must be Excised in a Case of Malignant Tumor at the Hepatico-cystic Juncture.

tion; and (2) Restoration of the continuity of the outlet, which may comprise end-to-end suture of the divided ends of the duct, or anastomosis of the upper segment of the duct with the intestine, and exclusion of the lower segment of the duct.

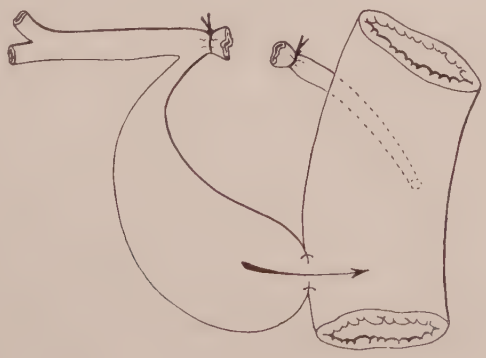

Fig. 10.—Resection of Choledochus, Closure of both Ends and Cholecystoduodenostomy. (*After Kehr.*)

In cases where the gall-bladder is healthy and the cystic duct patulous, we believe the operation of choice for stricture of the common duct is *cholecystenterostomy* (page 129); thus bile will be

discharged into the intestine in spite of the obstruction of the common duct.

Choledochoplasty of the supraduodenal choledochus, analogous to pyloroplasty, has been employed successfully by Petersen and by Moynihan, the latter employing temporary drainage of the duct. Stubenrauch has devised a series of plastic operations on the common duct, which are ingenious, but scarcely practicable. In one case he used with success a flap from the stomach to replace the common duct; Kehr employed a similiar operation without success, but in another case succeeded in saving his patient's life by employing a flap from the gall-bladder.

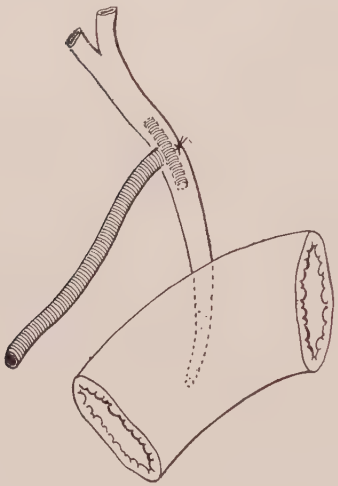

FIG. 11.—AFTER RESECTION OF CHOLEDOCHUS ITS POSTERIOR WALL HAS BEEN UNITED BY SUTURES AND A T-DRAINAGE TUBE INSERTED.

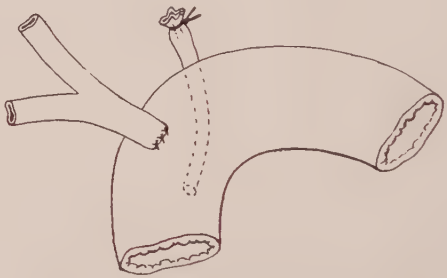

FIG. 12.—CHOLEDOCHO-ENTEROSTOMY. (*After Kehr.*)

Resection of the choledochus seldom has been done. Oppenheimer has collected eighteen operations, with an immediate mortality of 50 per cent.: ten operations were for carcinoma, with six deaths; four for cicatricial stricture, with two deaths; three for calculus, with no deaths; and there was one fatal operation for benign tumor. After resection of the choledochus end-to-end suture may be attempted; generally it will be sufficient to unite the posterior wall by two or three sutures, and leave a drainage-tube in the opening in the anterior wall. A T-drainage tube is useful in such cases, (Fig. 11,) and the defect in the choledochus should be covered with omentum. This supports the anastomosis, and the T-tube allows the bile to be discharged both into the duodenum and through the

abdominal wound. When granulations have had time to form, in the course of three to five weeks, the tube is removed through the abdominal wound, and the fistula allowed to close. Much more complicated methods have been used but not with notably better results.

If end-to-end suture of the resected common duct cannot be accomplished, the ends may be joined by a T-tube of rubber, and the gap reinforced by omentum,[1] as already suggested; or Sullivan's method may be employed (page 131). But if the obstruction is so close to the duodenal termination of the duct that

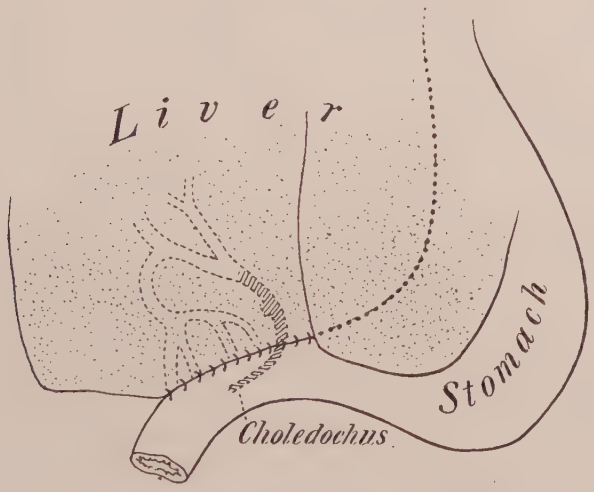

Fig. 13.—Diagram of Hepato-cholangeio-enterostomy. (*After Kehr.*)

resection cannot be done the duct may be divided above the stricture and its proximal end implanted into the duodenum, the distal end being closed (Fig. 12). This operation (*choledocho-enterostomy*) is less difficult when the duct has become dilated from long-standing back-pressure from a subjacent stricture than in ordinary cases. Termino-lateral implantation, suggested by Czerny in 1892, is preferable to the original lateral anastomosis employed by Riedel in 1888. If only the hepaticus is available for the anastomosis, *hepatico-enterostomy* may be attempted; it is advisible in such cases to resort to mobilization of the duodenum, and to fix it by sutures to the liver over the site of the anastomosis. Some

[1] Construction of artificial channels for the bile, by means of omentoplasty, was studied experimentally in 1901 by Enderlen and Justi.

anastomoses have also been made between dilated biliary radicles within the liver substance and the intestine; this operation is known as *hepatocholangeio-enterostomy*. It was proposed in 1896 by Baudouin, and in 1897 by Langenbuch and Ulmann. Monprofit in 1904 suggested the propriety of using a jejunal loop in-Y.

Though *transduodenal choledochotomy* for the removal of gallstones impacted at the ampulla of Vater has been done on numerous occasions, only a few such operations for stricture are on record (Körte, Oehler); and *excision* of an obstruction at this point seldom has been done except in the case of carcinomata (page 223).

While some form of cholecystenterostomy often is a very successful operation, the same cannot be said of the other procedures mentioned, as will be seen by the references tabulated at page 133. But in view of the extremely poor constitutional condition of the patients, the results are not surprising; and the surgeon certainly is justified in attempting to relieve an otherwise hopeless condition.

Choledochostomy, or union of the dilated common bile-duct with the abdominal wound has been done in a few rare instances, but is no longer a recognized operation. Usually the dilated duct has been mistaken for the gall-bladder itself. The term choledochostomy frequently is used synonymously with drainage of the choledochus. In the earliest operations on the common duct an attempt always was made to suture the incision made into it for the removal of a calculus; but this was soon abandoned,[1] and *choledochus drainage* is now an operation rather frequently performed in cases of cholangeitis, or after removal of a calculus by choledochotomy. References to the early operations on the common duct (by Kümmell, in 1884; by Thorton and by Heusner, in 1889 and by Courvoisier, in 1890) are given in Berger's monograph.

Hepaticostomy, or suture of the hepatic duct to the skin for the purpose of drainage has been done a few times under the same circumstances in which choledochostomy was formerly employed. According to Terrier, it was first done in 1889, by Kocher. In Bier's patient death occurred in seven days. In a patient under the care of Mayo the proximal end of the hepatic duct could not

[1] Kocher, however, in the last edition of his operative surgery, still advises its suture in all but exceptional cases.

be found after an extensive excision for carcinoma; all the bile was discharged by the wound, and death followed at the end of nine weeks. In the somewhat similar case of Kehr, death ensued in seven days from cholæmic hemorrhage. The term hepaticostomy sometimes is erroneously used as synonymous with *hepaticus drainage*. This, which is known also as *Kehr's operation* (1897), was employed as early as 1889 by Abbe, whose patient was still in good health when the operation was recorded, in 1893. It is practised quite frequently after choledochotomy, the drainage tube being passed upward past the entrance of the cystic duct, so as to drain the hepatic duct directly.

Hepatostomy, or drainage of the intrahepatic bile-passages, is indicated only when all the extrahepatic biliary ducts are strictured or occluded. First adopted by Sendler, in a case of suppurative cholangeitis, it was advocated by W. E. B. Davis and by Haasler. According to Berger this operation has been done by Hirschberg, whose technique consisted in performing hepatotomy by a large trocar, with subsequent dilatation and drainage of this channel. In some patients with chronic biliary obstruction, the dilated intrahepatic bile-channels project from the surface of the liver in the form of small cysts; and in most of these patients greatly dilated bile-spaces may be found close to the surface of the liver, even if they are not visible on its surface. So the operation is not as erratic as might seem to be the case at first thought. But the operation known as hepatocholangeio-enterostomy (page 138) should be preferred, when practicable. As Mathieu well says, the operation of hepatostomy is not one of choice, but is comparable to an enterostomy done for intestinal obstruction in the case of a patient too ill to endure the search for the site of occlusion; and, if the patient survives, a secondary operation will be necessary to restore the bile to the intestinal tract.

Cholecystenterostomy.—This operation was introduced in 1882 by von Winiwarter, who made the anastomosis with the colon (cholecysto-colostomy). Claudius H. Mastin (Mobile), and W. E. B. Davis (Birmingham), both of Alabama, were among the first in this country to do experimental work in connection with this subject. In recent times most surgeons have employed the duodenum (cholecysto-duodenostomy). Hildebrandt introduced the operation of cholecysto-gastrostomy, and it was employed by

Terrier as he found that the presence of the bile in the stomach produced no bad effects, and the anastomosis with the stomach usually was easier than with the duodenum. Kehr has adopted this operation, and according to Eichmeyer in a recent series of twenty-two anastomoses between the biliary tract and the gastro-intestinal tract Kehr adopted cholecysto-duodenostomy only once, and cholecysto-gastrostomy twelve times;[1] the other nine operations being as follows: choledocho-duodenostomy, two; hepatico-duodenostomy, four; hepatocholangeio-gastrostomy, one; hepato-cholangeio-cholecysto-gastrostomy, two. The jejunum may be used instead of the duodenum, as was done in former years not infrequently; but we believe the advantages of cholecysto-gastrostomy are now quite generally recognized.

In a patient with persistent biliary fistula, after cholecystostomy, Wilms connected this fistula by means of a rubber tube with a coil of the jejunum, the rubber tube passing across the skin from the fistula to another abdominal incision through which it entered the jejunum. Having thus proved that a cholecyst-enterostomy could be well borne by this patient, he performed a formal anastomosis between gall-bladder and intestine, with success.

For the purpose of preventing ascending infection from the jejunal loop, Krause employed an entero-anastomosis between the afferent and efferent loops of jejunum. Monprofit accomplished the same purpose by establishing a cholecysto-enterostomy in-Y, modelled on the gastro-jejunostomy in-Y of Roux of Lausanne (Vol. I, page 368). Kausch has employed a similar operation with success. Spannaus made the Y-anastomosis by the retrocolic route, following a suggestion of Brentano, but his patient died from pneumonia in twelve days. Krukenberg effected a torsion of 270 degrees in a long flabby gall-bladder, thus making it into a sort of cystic duct with spiral valve before anastomosing it with the intestine, in imitation of the principle introduced by Gersuny in operating for incontinence of fæces. None of these complicated methods is necessary if cholecysto-gastrostomy is employed; and in anastomosis with the duodenum the danger of ascending infection is very slight.

[1] Kehr has done in all over forty cholecysto-gastrostomies.

STATISTICS OF CHOLECYSTENTEROSTOMY.

Author.	Benign Lesions.		Malignant Lesions.		Total.		
	R.	D.	R.	D.	R.	D.	Mortality.
Bardeleben (1906)...	20	0	5	3	22	3	13.6 per cent.
Czerny { Merk, 1902 / Arnsperger, 1906. }	8	1	1	4	9	5	55.5 per cent.
Deaver (1910–1912).	16	0	4	0	20	0	.....
Körte (1905).....	3	4	7	3	10	7	70.0 per cent.
Robson (1904).....	17	1	2	5	19	6	31.5 per cent.

Hepatico-enterostomy by Means of a Rubber Tube.—When the distal end of the choledochus cannot be utilized in re-establishing the normal course of the bile it becomes necessary, when the gall-bladder is absent, to anastomose the proximal stump of the choledochus or the hepaticus with the duodenum. Occasionally this can be accomplished directly by suture; but often the duodenum, even after it has been mobilized, cannot be brought up near enough to the hepaticus to permit of this method being employed. The use of the jejunum in such circumstances is objectionable, but if an anastomosis can be made with the stomach, this should be done. Several surgeons have had the idea of reconstructing a channel for the bile from the hepaticus stump to the duodenum or stomach by means of a rubber tube. In some of the earlier operations it was thought necessary to withdraw this tube through the abdominal wound before this was permitted to close. Vollcker's technic is indicated in Fig. 14. Sullivan, however, working with Draper Maury, proved by experiments on dogs that it was safe to leave the tube in place, trusting to its expulsion into the duodenum and its final discharge from the rectum.

The plan of operation adopted by Sullivan may be summarized as follows: The tube used is approximately the size of the common duct; its duodenal end is tipped with a rubber or marine sponge not larger in diameter than half the lumen of the intestine. The proximal end of the choledochus or hepaticus is freely exposed, and after ligation is cut across just above the ligature. Two plain catgut sutures are introduced into one end of the tube, on opposite sides, and are then passed through the wall of the duct from within outward so as to draw the end of the tube into the open end of the duct when the sutures are drawn taut. The tube is carried into the lumen of the duct about one centi-

metre. The other end of the tube, carrying the sponge, is then implanted into the anterior wall of the duodenum at the level of the papilla of Vater by means of a Witzel fistula (Vol. I, page 354). The tube itself is sutured to the duodenum at one point with fine catgut so as to prevent its too early expulsion into the intestinal canal. The great omentum is then drawn up and a suitable area is traumatized lightly with gauze friction; similar friction is applied to the duodenum and gastrohepatic omentum on both

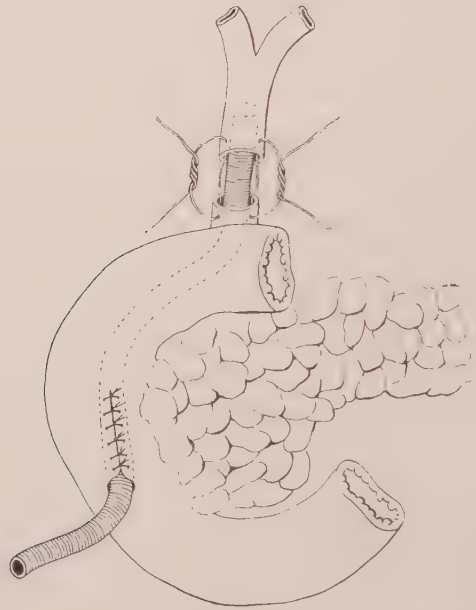

FIG. 14.—VOELCKER'S METHOD OF CHOLEDOCHO-(HEPATICO)-DUODENOSTOMY BY MEANS OF A RUBBER TUBE WHICH EMERGES FROM THE DUODENUM THROUGH A WITZEL FISTULA.

sides of the tube. The great omentum is then adjusted so as to cover the tube and extend beyond it in all directions, and is held in place by several fine catgut sutures. After the retaining sutures of catgut have been absorbed the tube is drawn into the intestine by the tug on the sponge tip exerted by intestinal peristalsis. A more or less permanent channel is formed in this way, permitting the discharge of bile into the intestine.

Brewer has adopted a similar operation with success in one case; and Wilms has employed it successfully in three cases. In

several instances the tube never was recovered from the fæces, but as it could not be detected by the X-rays it was presumed to have passed unnoticed. Brandt thinks it desirable that it should remain permanently *in situ*.

STATISTICS OF END-TO-END SUTURE OF CHOLEDOCHUS AFTER RESECTION.

Operator.	Lesion.	Result.
Doberauer (Beitr. z. klin. Chir., 1910, lxvii, 472).	Carcinoma.	Died in 3 mos. (Suture of posterior wall and drainage.)
Doyen (Arch. Prov. de Chir., 1892, i, 169).	Operative injury.	Died in 8 days of cholæmia. (Suture over rubber tube left *in situ*.)
Jaboulay (Patel: Lyon Méd., 1903, ci, 1002).	Carcinoma.	Died 4 days. (See Brandt: Deutsch. Zeit. f. Chir., 1912, cxix, 1.)
Kehr (1899) (Technik d. Gallenstein-op., Münich 1905; Fall 151).	Cicatricial stricture.	Recovered, and well after 5 weeks.
Kehr (1907) (Eichmeyer: Arch. f. klin. Chir., 1911, xciv, 1; Fall 127).	Postoperative stricture.	Recovered and well after 3 mos. (Sutures reinforced by flap from gall-bladder.)
Kehr (1908) (Eichmeyer: loc. cit., Fall 128 and 134).	Postoperative defect, with fistula.	Recovered and well after 5 weeks.
Kehr (1909) (Eichmeyer: loc. cit., Fall 125 and 130).	Postoperative stricture.	Recovered and well after 6 weeks. (Drained by T-tube.)
Kehr (1908) Kehr, Liebold u. Neuling, Drei Iahre, u. s. w., S. 404, Fall 229.	Two months after operative injury.	Recovered and well after 5 weeks. (End-to-end suture, duct not drained.)
Körte (Verh. d. Deutsch. Ges. f. Chir., 1903, xxxii, 626; Chir. d. Gallenwegen, Berlin, 1906, S. 341, Fall 208).	Cicatricial stricture.	Died 13 days. (Hemorrhage from gastric ulcer.) Also did cholecysto-duodenostomy.
Mayo (Collected Papers, previous to 1909; Phila. 1912, vol. i, p. 446).	Operative injury.	Recovered and well after 2 years.
Mayo (Ibid.).	Carcinoma.	Recovered, but died of recurrence in 13 mos.
Mayo (Ibid.).	Carcinoma.	Died 3 days.
Moschowitz (Annals of Surgery, 1912, i, 610).	Carcinoma.	Recovered but biliary fistula persisted after 5 weeks.
Moynihan (Gall-stones, Phila., 1905, p. 433).	Carcinoma.	Died from shock in few hours.
Moynihan (Abdominal Operations, Phila. 1906, p. 690).	Carcinoma.	Recovered, but died of recurrence in 3 mos.

Riese (Verh. d. freien Vereinigung d. Chir. Berlins, 1905, xvii, ii, 52).	Carcinoma.	Died.
Rotter (Volmer: Arch. f. klin. Chir., 1908, lxxxvi, 160).	Adenomyofibroma.	Died 3 days.

STATISTICS OF CHOLEDOCHO-ENTEROSTOMY.

Operator.	Lesion.	Result.
Brin (Bull. Soc. Chir. Paris, 1905, xxxi, 945).	Cicatricial stricture.	Recovered and well after 8 mos.
Czerny (1902) (Arnsperger: Beitr. z. klin. Chir., 1906, xlviii, 673, Fall 10).	Postoperative stricture.	Recovered, but small fæcal fistula persisted 1 year later.
Czerny (1903) (Ibid., Fall 23).	Carcinoma.	Died 2 days. (Also posterior gastro-enterostomy.)
Fullerton (Brit. Med. Jour., 1907, ii, 1118).	Stricture from pancreatic disease.	Recovered and well after 5 mos. (Used Murphy button.)
Hartmann (Bull. Soc. Chir. Paris, 1905, xxxi, 1104; Mathieu: Rev. de Chir., 1908, i, 65).	Congenital obstruction.	Recovered. Operation at age of 3 1/2 years.
Kausch (Zentr. f. Chir., 1911, xxxviii, 159; Verh. d. Deutsch. Gesell. f. Chir., 1911, xl, i, 290).	Undetermined obstruction at Papilla of Vater.	Recovered and well after 1 year. (Choledochojejunostomy in-Y, extraperitoneal.)
Kehr (Gall-stone Dis., Phila., 1901, p. 281).	Chronic pancreatitis.	Recovered.
Kehr (Ibid., p. 184).	Carcinoma.	Died 13 days.
Kehr (Eichmeyer: Arch. f. klin. Chir., 1910, xciii, 887; Fall 148).	Chronic pancreatitis.	Recovered and well after 1 year.
Kehr (Ibid., Fall 149).	Cicatricial stricture.	Recovered and well after 3 weeks.
Marwedel (Arnsperger: Beitr. z. klin. Chir., 1906, xlviii, 698, Fall 15).	Cicatricial stricture.	Recovered and well after 4 1/2 years.
Mayo (Collected Papers, previous to 1909, Phila., 1912, vol. i, p. 447).	Carcinoma.	Died 8 weeks.
Mayo (Ibid.).	Carcinoma.	Died 9 weeks.
Mayo (Ibid.).	Carcinoma.	Recovered.
Riedel (1888) (Erfahrungen u. d. Gallensteinkrankh., Berlin, 1892, S. 117).	Calculous obstruction.	Died 1 day. (Designed an end-to-side implantation but had to do lateral anastomosis.)
Rowlands (Jacobson and Rowlands, Ops. of Surgery, Phila., 1908, ii, 563).	Obstruction by pancreatic cyst.	Result not recorded.
Rosemberger (Deutsch. Zeit. f. Chir., 1906, lxxxv, 275).	Cicatricial stricture.	Recovered and well after 10 mos.

Robson (1892) (Dis. of Gall-bladder and Bile-ducts, London, 1897).	Cicatrical stricture.	Recovered and well in 6 mos. Then choledochotomy for obstruction by stone, and death in two weeks.
Robson (1895) (Dis. of Gall-bladder and Bile-ducts, 3d. Ed., London, 1904, p. 314, Case 121).	Cicatricial stricture.	Recovered and well after 1 year. (Op. was cystico-duodenostomy, not choledocho-duodenostomy.)
Robson (Ibid., p. 197, Case 526).	Chronic pancreatitis.	Recovered and well 2 mos.
Souligoux (Bull. Soc. Chir. Paris, 1907, xxxiii, 990).	Pancreatic obstruction.	Recovered and well over 3 mos.
Sprengel (Verh. d. Deutsch. Ges. f. Chir. 1891, xx, 132).	Cicatricial stricture.	Recovered and well after 6 mos. (Lateral anastomosis.)
Summers (Jour. Amer. Med. Assoc., 1900, i, 592).	Cicatricial stricture.	Recovered and well after 3 mos. (Used Murphy button.)
Swain (Lancet, 1895, i, 743).	Stricture.	Recovered and well after 2 mos. (Used Murphy button.)
Tédenat (1902) (Assoc. Franç. de Chir., 1908, xxi, 189; Zentr. f. Chir., 1909, xxxvi, 763).	Papilloma.	Recovered, and well after 6 years.

STATISTICS OF HEPATICO-ENTEROSTOMY.

Operator.	Lesion.	Result.
Dahl (Zentr. f. Chir., 1909, xxxvi, 266).	Post operative stricture.	Recovered and well after 8 mos. (Op. by retrocolic hepatico-jejunostomy in-Y in May, 1908, or 5 mos. before this technique was suggested by Monprofit.)
Delbet (Bull. Soc. Chir. Paris, 1907, xxxiii, 949).	Pancreatic Tumor.	Recovered and well 3 weeks.
Enderlen (Oppenheimer: Deutsch. Zeit. f. Chir., 1912, cxv, 415).	Carcinoma.	Recovered and well after 1 year.
Enderlen (Münch. med. Woch., 1908, ii, 2066).	Postoperative stricture.	Died 8 days, from internal hemorrhage.
Kehr (Verh. d. Deutsch. Ges. f. Chir., 1904, xxxiii, 65; Kehr's Technik, Fall 152; Eichmeyer: Arch. f. klin. Chir., 1911, xciv, 12).	Carcinoma.	Recovered; died after 2 1/2 years of hepatic abscess.
Kehr (Eichmeyer: Arch. f. klin. Chir., 1910, xciii, 888, Fall 150.)	Carcinoma of pancreas and cholelithiasis	Died 3 days. (Cholæmia and uræmia.)
Kehr (Eichmeyer, loc. cit., 1910, xciii, 888, Fall 151).	Postoperative stricture.	Recovered and well after 3 mos.

Kehr (Eichmeyer, loc. cit., 1910, xciii, 888, Fall 152).	Postoperative stricture.	Recovered and well for 6 mos; then recurrence and another operation.
Kehr (Eichmeyer, loc. cit., 1910, xciii, 888, Fall 153, same patient as Fall 152).	Postoperative stricture (recurrent).	Died 3 days.
Lejars (Semaine Méd., 1911, xxxi, 37).	Carcinoma.	Recovered.
Lanphear (Surg., Gyn. and Obst., 1909, i, 406).	External biliary fistula, following drainage of pericholecystic abscess.	Recovered; no after history. (Retrocolic hepaticojejunostomy in-Y.)
Quénu (Bull. Soc. Chir. Paris, 1905, xxxi, 218). Operation in 1903.	Cicatricial stricture.	Died 2 days. (Hepaticogastrostomy.)
Terrier (Bull. Soc. Chir. Paris, 1907, xxxiii, 17).	Postoperative stricture.	Recovered and well 2 mos.
Terrier (Ibid.).	Obstruction by pancreatic lesion.	Recovered and well 1 mo.
Terrier (Mathieu: Rev de Chir., 1908, xxxvii, 204).	Cicatricial stricture, recurrence after operation to form new common duct by rubber tubage and omentoplasty.	Recovered and well after 1 month.
Tuffier (Bull. Soc. Chir. Paris, 1905, xxxi, 251).	Carcinoma.	Died in 2 months. (Hepatico-gastrostomy).
Tuffier (Bull. Soc. Chir. Paris, 1907, xxxiii, 30.)	Tumor.	Died 36 hours.

STATISTICS OF HEPATICO-ENTEROSTOMY BY RUBBER TUBAGE.

(Where Ends could not be Approximated, after Excision of Choledochus.)

Operator.	Lesion.	Result.
Brewer (Annals of Surg., 1910, i, 830).	Sloughing after gangrene of gall-bladder.	Recovered and well after 7 weeks. Tube left to be passed by rectum.
Jenckel (Deutsch. Zeit. f. Chir., 1910, civ, 40. Operation Nov. 25, 1905).	Cicatricial stricture.	Recovered and well over four years. Tube from hepaticus implanted into duodenum by Witzel fistula, and removed through abdominal wound later.
Terrier (1906) (Bull. Soc. Chir. Paris, 1907, xxxiii, 17; Mathieu: Rev. de Chir., 1908, xxxvii, 203).	Postoperative stricture.	Recovered. (Op. by tube in hepaticus stump, another in choledochus stump, both emerging from abdominal wound; common duct con-

		structed around them by omentoplasty.) Four months later did hepatico-enterostomy by suture, as there was no permanent improvement.
Verhoogen (Congr. Franç. de Chir., 1908, xxi, 201; de Graeuwe: Zentralbl. f. Chir., 1908, xxxv, 790; Op. in 1907).	Not stated.	Recovered, and well after 16 mos. (Single T-tube inserted in stump of choledochus and brought out of abdominal wound. Large window at angle of tube next stump of hepaticus.)
Voelcker (Beitr. z. klin. Chir., 1911, lxxii, 581).	Twenty-four days after op. injury (carcinoma) causing complete external fistula.	Recovered and went home on 16th day. (Tube from hepaticus implanted into duodenum, and brought out of duodenum at lower point through Witzel fitsula, which was sutured to skin. Tube removed on 5th day.)
Voelcker (Ibid., S. 593).	Six days after operative injury. (A colleague's operation.)	Recovered, but worse external biliary fistula developed. (Rubber tube left *in situ*.)
Voelcker (Ibid., S. 593, same patient as last).	For external biliary fistula, 5 mos. after op. injury.	Recovered and went home on 20th day. (Found tube left at first op. still in common duct and obstructing it. Implanted tube as in first of 3 ops. here detailed.)
Wilms (Leuenberger: Corr.-Bl. f. Schw. Aerzte, 1910, xl, 737; Zentr. f. Chir., 1911, xxxiii, 1506; Brandt: Deut. Zeit. f. Chir., 1912, cxix, 8).	Carcinoma.	Recovered, but biliary fistula persists. (Tube sutured in jejunum by Witzel, and left *in situ*.)
Wilms (Brandt: Deut. Zeit. f. Chir., 1912, cxix, 1, Fall 1).	Calculous obstruction with stricture or pancreatitis.	Recovered and well 15 mos. (Tube from hepaticus to duodenum never removed).
Wilms (Brandt, ibid., Fall 2).	Cicatricial stricture.	Recovered and well 15 mos. (Tube from hepaticus to duodenum, removed through abdominal wound.)
Wilms (Brandt, ibid., Fall 3).	Cicatricial stricture.	Recovered and well 8 mos. Tube left to be passed as in Brewer's method; but had to be removed through abd. wd., was then replaced, and later was vomited.

Wilms (Brandt, ibid., Fall 4).	Postoperative biliary fistula, from injury to choledochus.	Recovered; well 6 mos. after last op. (First op. created defect of choledochus; 2nd op. tube from hepaticus to choledochus stump, result biliary fistula; 3rd op. removed tube (kinked) and did Voelcker's op. as above; recurrence, therefore 4th op. tube from hepaticus to stomach, this was vomited after 3 mos.; therefore 5th op., for renewed obstruction, same technic as 4th op. Recovered and well 6 mos.)
Wilms (Brandt, ibid., S. 19, Fall 5).	Obstruction at papilla of Vater.	Recovered. (Tube from lateral incision in choledochus implanted in duodenum by Witzel fistula. Tube remains *in situ*.)

STATISTICS OF HEPATO-CHOLANGEIO-ENTEROSTOMY.

Operator.	Lesion.	Result.
Czerny (1898) (Merk: Mitth. a. d. Grenzgeb. d. Med. u. Chir., 1902, ix, 566, Fall 111).	Carcinoma.	Died 2 days. (Operation done 5 weeks after cholecystenterostomy.)
Doberer (Wien. klin. Woch., 1910, xxiii, 1445).	Postoperative stricture.	Recovered and well after 2 mos.
Ehrhardt (Zentr. f. Chir., 1907, xxxiv, 1226).	Congenital aplasia of bile-ducts; op. at age of 6 weeks.	Died at end of 8 days, from enteritis. (Jejunum sutured to liver.)
Garrè (Beitr. z. Phys. u. Path. (Weiss), Stuttgart 1908, S. 22; Rev. de Chir., 1908, xxxviii, 571).	For traumatic stricture of hepaticus (this had been drained for rupture, 7 weeks after original injury).	Recovered and well 3 1/2 years. (Duodenum was sutured to liver 18 mos. after original injury, because obstruction recurred, after original hepaticus drainage.
Jordan (Verh. d. Deutsch. Ges. f. Chir., 1899, xxviii, i, 153).	Carcinoma.	Died. At previous op. had done cholecystenterostomy; but biliary fistula formed in a tear in liver, and finally fæces came by reflux through liver fistula (up gall-bladder to cystic duct, through hepaticus to liver). Therefore did anastomosis

		between intestine and biliary fistula in liver, but patient died from peritonitis.
Kehr (Verh. d. Deutsch. Ges. f. Chir., 1904, xxxiii, 65; Zentr. f. Chir., 1904, xxxi, 185).	Obstruction from adhesions and pyloric ulcer.	Recovered, but died in 2 mos.
Lameris (Zentr. f. Chir., 1912, xxxix, 1665).	Carcinoma.	Recovered, but died of recurrence in 8 mos.
Lejars (Semaine Méd., 1909, xxix, 121).	Cicatricial stricture of hepaticus.	Died 4 days, of liver infection.
Maylard (Annals of Surg., 1905, i, 56).	Adhesions.	Died 1 month.

Biliary Obstruction from Causes Outside of the Ducts.—The most frequent of these causes is the presence of pericholecystic adhesions (page 53), which cause kinking of the cystic or common duct. The symptoms and treatment of this condition have been considered at page 98.

Obstruction from diseases of the pancreas is discussed at page 356.

Obstruction from carcinoma of the papilla of Vater is not very rare (page 223).

Obstruction from kinking of the ducts due to the presence of a *movable kidney* was first recognized by Weissker, in 1888, and has been especially studied by Tinker. According to Tinker, Apolant believes that many of the patients treated at Carlsbad for supposed affections of the biliary tract are in reality suffering from pressure of a misplaced kidney. Tinker reports two cases of his own in which the patients secured immediate and permanent relief of all symptoms referable to the biliary tract after the operation of nephropexy. The *symptoms* presented are those of interference with the normal flow of bile, indigestion, nausea and occasionally vomiting, pain in the right hypochondrium referred to the back or shoulder, and in some cases intermittent jaundice. Although the symptoms all point to the biliary tract, they are relieved by the return of the kidney to its normal position, either by manipulation, or when the patient assumes the recumbent position.

The *diagnosis* usually is not made until operation has demonstrated that there are no lesions in the biliary tract. The possi-

bility of a movable kidney as a cause should be borne in mind, and in cases of doubt this factor should be eliminated by rest in bed or by the use of a well-fitting corset with a kidney pad, before operation is undertaken. When it has been ascertained definitely that a movable kidney is the cause of the biliary symptoms, the operation of nephropexy should be advised.

Obstruction from Aneurism.—This is a rare cause of biliary obstruction. According to Villandre, most cases have developed shortly after some acute infection, especially pneumonia or typhoid fever. The aneurism may affect the abdominal aorta, or the hepatic artery or one of its branches. The symptoms to attract attention usually have been those of pressure, and not any symptoms attributable to the aneurism as such. Bode in 1909 collected thirty-eight cases of aneurism of the hepatic artery. In most cases the diagnosis has been made only at autopsy. Villandre thinks that if the triad of symptoms (local pain, intestinal hemorrhages, and jaundice) is present, the existence of an aneurism of the hepatic artery should be suspected. These are the most characteristic symptoms, but one or more often is absent. Intestinal hemorrhage alone may be attributed to duodenal ulcer, and even when associated with local pain and jaundice might be due to malignant obstruction of the common duct.

Villandre has collected the following cases of aneurism of the hepatic artery in which operation was done:

Mikulicz: lesion not found; gastro-enterostomy. Death in six days. Diagnosis at autopsy.

Riedel: incised aneurism, thinking it was calculus in common duct; tampon for hemorrhage. Second operation, twenty days later, abandoned and tamponed for hemorrhage. Death in three days. Diagnosis at autopsy.

Heller: found blood in abdominal cavity, mesocolon, and in gall-bladder and cystic duct. Tamponed. Death.

Habs (report by Grunert): after cholecystectomy recognized aneurism of hepatic artery as cause of choledochus obstruction. Nothing further done. Death in eight days.

Allessandri: cholecystotomy; hemorrhage; tampon. Death in five days.

Tuffier: incised mass in gastro-hepatic omentum; profuse hemorrhage; ligated hepatic artery. Death in four days.

Kehr: incised gall-bladder; gush of blood, controlled only by ligation of hepatic artery. The gall-bladder was then extirpated and sac of aneurism packed. Aneurism had ruptured into gall-bladder and further hemorrhage

prevented only by plugging of cystic duct by clot. Recovered, and according to Bode in good health six years later.

To these may be added an operation by Garrè, reported by Bode: over a year after an injury to the right thorax Garrè operated on a patient who presented symptoms of pyloric obstruction with gastro-intestinal hemorrhages. The diagnosis was gastric ulcer, and gastro-enterostomy was done. The patient died a week later, and at autopsy an aneurism was found on an intrahepatic branch of the hepatic artery; this had ruptured into one of the intrahepatic bile-ducts, but there had been no recent hemorrhages. There were scars of old injury in the liver and right kidney.

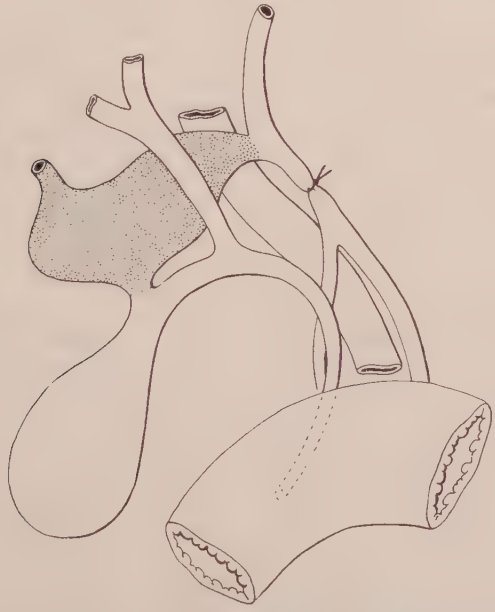

FIG. 15.—KEHR'S CASE OF ANEURISM OF THE HEPATIC ARTERY WHICH HAD RUPTURED INTO THE GALL-BLADDER. LIGATURE ON THE HEPATIC ARTERY.

Rational operation (ligation of the hepatic artery) was done only in two cases, those of Tuffier and Kehr.

Kehr's is the only patient who recovered. Villandre conducted experiments on dogs, to ascertain the feasibility of survival after ligation of the hepatic artery. Though few in number these experiments tended to demonstrate that while gradual occlusion is safe, yet rapid occulsion, such as occurs in embolus or after ligation of the normal artery, always is fatal. Evidently in Kehr's case adequate collateral circulation had been established

before the operation. It is noteworthy that Kehr has also successfully ligated the hepatic artery in one case for hemorrhage during choledochotomy.

Körte did cholecyst-enterostomy for obstruction from a mass at the papilla of Vater (probably a congenital stricture); death occurred from internal hemorrhage, and at autopsy a thrombosed aneurism was found in the course of the bile-ducts. This had not caused any symptoms.

Portal thrombosis, according to Bode, has been treated by operative means in eight cases, only three patients recovering—two after the Talma operation for ascites from cirrhosis of the liver (epiplopexy), and one after simple drainage of the ascitic fluid.

REFERENCES.

Abbe: Medical Record, New York, 1893, i, 548.
Arnsperger: Beitr. z. klin. Chir., 1906, xlviii, 673.
Bardeleben: Erfahrungen über Cholecystektomie, Jena, 1906, S. 46.
Baudouin: Progrès Méd., 1896, xxiv, 257.
Baudouin: Les Operations Nouvelles sur les Voies Biliaires, Paris,, 1897.[1]
Berger: Arch. f. klin. Chir., 1903, lxix, 298.
Bier: quoted by Oppenheimer, loc. cit. (Hepaticostomy).
Bode: Beitr. z. klin. Chir., 1909, lxiv, 505 (Portal thrombosis).
Bode: Ibid., S. 516 (Aneurism).
Brandt: Deutsch. Zeit. f. Chir., 1912, cxix, 1.
Brentano: Zentr. f. Chir., 1907, xxxiv, 681.
Brewer: Annals of Surgery, 1910, i, 830.
Coffey: Annals of Surgery, 1909, ii, 1238.
Cordier: Surg., Gyn. and Obst., 1906, ii, 380.
Czerny: for statistics of cholecystenterostomy see Arnsperger and Merk.
Davis, W. E. B.: Jour. Amer. Med. Assoc., 1901, ii, 1573.
Eichmeyer: Arch. f. klin. Chir., 1910, xciii, 857; 1911, xciv, 1.
Enderlen and Justi: Deutsch. Zeit. f. Chir., 1901, lxi, 235.
Enderlen: Verhand. d. Deutsch. Ges. f. Chir., 1904, xxxiii, 77.
Enderlen and Zumstein: Mitth. a. d. Grenzgeb. d. Med. u. Chir., 1904, xiv, 104 (experimental hepato-cholangeio-enterostomy).
Gaston: Atlanta Med. and Surg. Jour., 1884, xxv, 385 (experimental cholecystenterostomy).
Grunert: Deutsch. Zeit. f. Chir., 1904, lxxi, 158.
Haasler: Zentr. f. Chir., 1904, xxxi, 1366 (experimental hepato-cholangeio-enterostomy).
Hartmann: Bull. Soc. Chir., Paris 1905, xxxi, 1106.

[1] This work has not been accessible to us.

Hildebrandt: Deutsch. Zeitr. f. Chir., 1902, lxvi, 379.
Kausch: Zentr. f. Chir., 1909, xxxvi, 1350: 1911, xxxviii, 100 (circular resection of duodenum for carcinoma of papilla of Vater; death in 9 mos. of cholangeitis).
Kehr: Münch. med. Woch., 1897, xliv, 1127 (Hepaticus drainage).
Kehr: Verh. d. Deutsch. Ges. f. Chir., 1904, xxxiii, 65 (Aneurism).
Kehr: Technik der Gallenstein-Operationen, Münich, 1905.
Kehr, Leibold u. Neuling: Drei Jahre d. Gallenstein chir. München, 1908, S. 404, Fall 229.
Kehr: case of hepaticostomy; see Eichmeyer, loc. cit., 1910, xciii, 857, Fall 129.
Körte: Verhandl. d. Deutsch. Ges. f. Chir., 1903, xxxii, 622 (transduodenal choledochotomy for stricture).
Körte: Beitr. z. Chir. d. Gallenwegen. u. d. Leber, Berlin, 1906 (Cholecystenterostomy, S. 223; aneurism, S. 225; resection of common duct, S. 341).
Krause: see Maragliano.
Krukenberg: Zentr. f. Chir., 1904, xxxi, 125.
Langenbuch: Chir. d. Leber u. Gallenblase, Stuttgart, 1897, ii, 357 (hepatocholangeio-enterostomy).
Lejars: Semaine Méd., 1911, xxxi, 37.
McBurney: N. Y. Med. Jour., 1891, liii, 520.
McBurney: Annals of Surgery, 1898, xxviii, 481, Case 2 (transduodenal choledochotomy in 1891).
Maragliano: Zentr. f. Chir., 1903, xxx, 941.
Mathieu: Revue de Chir., 1908, xxxvii, 61, 174.
Merk: Mitth. a. d. Grenzgeb. d. Med. u. Chir., 1902, ix, 540.
Monprofit: Semaine Méd., 1904, xxiv, 333.
Moynihan: Brit. Med. Jour., 1905, ii, 380.
Oehler: quoted by Oppenheimer, loc. cit.
Oppenheimer: Deutsch. Zeit. f. Chir., 1912, cxv, 415.
Parkes: Trans. Amer. Surg. Assoc., 1885, iii, 335.
Petersen: Verhandl. d. Deutsch. Gesellsch. f. Chir., 1904, xxxiii, 80 (Czerny's case of hepato-cholangeio-enterostomy.)
Petersen: transduodenal choledochotomy for stricture, quoted by Arnsperger, loc. cit., Fall 14.
Riedel: Erfahrungen über die Gallensteinkrankheit. Berlin, 1892, S. 117.
Robson: Dis. of Gall-bladder and Bile-ducts, 3d Ed., London, 1904, p. 313. (cholecystenterostomy).
Schüller: Beitr. z. klin. Chir., 1901, xxxi, 683 (Czerny's case of cholecystenterostomy for carcinoma; died in 8 days).
Sendler: Deutsch. Zeit. f. Chir., 1895, xl, 405.
Sims, J. Marion: Brit. Med. Jour., 1878, i, 811.
Spannaus: Beitr. z. klin. Chir., 1910, lxx, 604.
Sprengel: Verh. d. Deutsch. Ges. f. Chir., 1891, xx, 132.
Stubenrauch: Verh. d. Deutsch. Ges. f. Chir., 1906, xxxv, ii, 39.
Sullivan: Jour. Amer. Med. Assoc., 1909, ii, 776.

Terrier and Auvray: Chir. du Foie et des Voies Biliaires, Paris, 1901–1907.
Tinker: Jour. Amer. Med. Assoc., 1907, ii, 124.
Ulmann: Wien. med. Woch., 1897, xlvii, 2282.
Villandre: Arch. Gén. de Chir., 1909, iv, 31,111.
Weissker: Schmidt's Jarhb., 1888, ccxx, 249.
Wilms: quoted by Brandt, loc. cit. supra.
v. Winiwarter: Prag. med. Woch., 1882, vii, 201, 213 (cholecystenterostomy, recovered).

Biliary Fistula.

A **biliary fistula** is one that discharges, or has discharged bile. Such fistulæ are not uncommon. They result in most cases at the present day from operations on the biliary tract; but they may also be caused by inflammatory changes with perforation, or from carcinoma of the gall-bladder or bile-ducts. They are classified as *internal* or *external*. External fistulæ are those which open upon the surface of the body; internal fistulæ are those which communicate with one of the internal organs.

Courvoisier collected the following statistics in regard to the location of biliary fistulæ in 499 cases.

COURVOISIER'S STATISTICS OF BILIARY FISTULA.

External fistulæ	196 cases.	
Internal fistulæ	303 cases	
Between biliary tract and peritoneal cavity	70	127
Between biliary tract and peritoneal adhesions	49	
Between biliary tract and retroperitoneal tissues	3	
Between biliary tract and portal vein	5	
Between biliary tract and thoracic organs	24	24
Between biliary tract and urinary organs	7	7
Between biliary tract and other abdominal viscera:		
Stomach	13	137
Duodenum	83	
Jejunum	1	
Ileum	1	
Colon	39	
Between biliary tract and other portions of biliary tract	8	8
Total internal fistulæ		303

Internal Biliary Fistula.—A fistulous opening between the biliary tract and one of the adjacent viscera may occur after an

adhesive peritonitis has bound the two structures together; this forms the *direct* variety of internal fistula. If the communication occurs through an abscess cavity, the affected viscera not being in direct contact, the fistula is said to be of the *indirect* variety. As a rule, perforations of the biliary tract into the free peritoneal cavity or into peritoneal adhesions are not classed as biliary fistulæ, though they are included in Courvoisier's statistics quoted above.

A true fistula may exist between two or more of the biliary passages themselves; between the biliary passages and the substance of the liver or pancreas; or the fistula may join the gall-bladder or ducts with the lumen of the stomach, duodenum, colon, or some portion of the small intestine. There are on record a number of cases of biliary fistulæ communicating with the urinary, thoracic (pulmonary, pleural, pericardial, mediastinal), and female pelvic organs, but these are pathological curiosities and have comparatively little interest for the surgeon. Usually they have been found at autopsy, or have developed so soon before death that no attempt at operative relief has been justifiable. They are much rarer now than in the period before diseases of the biliary tract came under the domain of surgery.

Symptoms.—When the biliary fistula is of such size as to permit the discharge from the gall-bladder of all its contained calculi, with free drainage of the infected biliary tract, all inflammatory symptoms quickly subside. Nature's cholecystenterostomy often is as successful as an operative anastomosis in relieving the patient of distressing symptoms. The true condition of affairs may be surmised, if a patient passes by rectum one or more calculi too large to have traversed the bile-ducts; in one case on record the diagnosis was made from the fact that the patient vomited gall-stones. In this case recovery ensued without operation.

In the following case, which came under the senior author's care a number of years ago, a cholecysto-gastric fistula was found but there was no history of the passage or vomiting of gall-stones, and none were found at operation :

K. H., female, aged forty-nine; admitted to the German Hospital June 22, 1903. Has complained of present trouble for last ten years. Intermittent attacks of sharp abdominal pain, accompanied by vomiting. No jaundice at any time. For last three weeks has had severe abdominal pains, especially

marked in epigastrium and right hypochondrium, with vomiting and "biliary eructations" of gas.

Examination.—Tenderness in epigastrium and over gall-bladder. Slight rigidity of upper right rectus. Mitral systolic murmur. Has had simple goiter since girlhood. Liver dullness normal.

Operation by Dr. Deaver. Ether anæsthesia. Incision through upper right rectus. Adhesions between gall-bladder, ducts, stomach, duodenum, and omentum. Gall-bladder soft and contracted. Gall-bladder firmly attached to the stomach into which there was a fistulous opening from the gall-bladder. Adhesions were broken up, the opening in the stomach closed, the gall-bladder was removed, and the common duct drained. No biliary calculi could be found. Patient recovered.

The three following cases represent progressive stages in the development of internal biliary fistulæ:

Pericholecystitis; Calculi among Adhesions around Gall-bladder, Cholecystostomy. Recovery.

R. Z., female, aged thirty years; admitted to the German Hospital November 22, 1906. Mother died of carcinoma of the liver. Patient had typhoid fever when twenty years old. Bowels always constipated. For fifteen years has had attacks of pain in epigastric and gall-bladder regions, with vomiting. No jaundice until attack four months ago, when she had jaundice with chills and fever.

Examination.—No jaundice on admission. Liver normal; gall-bladder palpable. Tenderness and rigidity in gall-bladder area. W. B. C., 10,000.

Operation by Dr. Deaver. Ether anæsthesia. Incision splitting fibres of upper right rectus. Adhesions around gall-bladder, and between it and the omentum. On the outside of the wall of the gall-bladder were several gall-stones which seemed to have worked their way gradually through the walls of this viscus. They seemed to be very lightly attached to the gall-bladder and fell off during light manipulation. The gall-bladder was distended and packed with stones. Stones removed and gall-bladder drained with rubber tube. Recovery.

Cholelithiasis; Cholecysto-gastric Fistula. Cholecystostomy; Repair of Stomach. Recovery.

Mrs. F., aged fifty-three years; admitted to the German Hospital November 22, 1907. Was admitted as an eye patient. While in hospital had a typical attack of biliary colic which was a repetition of many previous attacks.

Operation by Dr. Deaver. Ether anæsthesia. Incision splitting fibres of upper right rectus, and extended to ensiform along costal border. On opening peritoneum dense adhesions were seen below liver margin completely hiding gall-bladder. Right lobe of liver smaller than normal. Large and small gauze pads introduced to isolate operative field. Marine sponges

introduced just below gall-bladder area. Adhesions divided. In freeing stomach, which was adherent to gall-bladder, a small hole was found in stomach. Examining gall-bladder at point where it had been adherent to stomach, a small opening was found also so that a cholecysto-gastric fistula must have been formed spontaneously prior to operation. Gastric opening, which was size of goose-quill, closed by purse-string suture of linen. Gall-bladder opening enlarged and stone (2.5 by 2.5 cm.) removed from gall-bladder. Gall-bladder wall very thick. Gall-bladder drained with rubber tube, and cigarette drain to subhepatic pouch. Recovery.

Cholelithiasis; Cholecysto-gastric Fistula. Cholecystectomy; Repair of Stomach; Drainage of Choledochus. Recovery.

Mrs. W. D., aged 42 years; admitted to the German Hospital April 10, 1911. Has had numerous attacks of biliary colic, extending over many years. Has made several trips to Carlsbad, always with temporary improvement. Operation undertaken because of persistent recurrence of attacks.

Operation by Dr. Deaver. Ether anæsthesia, preceded by nitrous oxide. Incision splitting fibres of upper right rectus, extended inward along costal margin. Duodenum and pylorus bound by strong adhesions to fundus of gall-bladder and lower surface of liver. On releasing adhesions a perforation of stomach and of gall-bladder was found, with a large gall-stone protruding into stomach. Adhesions freed, and opening in stomach closed. The free border of gastro-hepatic omentum was next opened, exposing cystic and common ducts. Cystic duct and artery ligated and cut, and gall-bladder removed, after calculus had been pushed back into gall-bladder. Gall-bladder was thickened and contracted. A large stone, the size of end of thumb, occupied gall-bladder cavity. Mucosa of gall-bladder œdematous and ulcerated. Margin of cystic duct grasped with hæmostat and probe passed down common duct to duodenum; hepatic ducts also free from obstruction. Small rubber tube sutured into cystic duct opening with chromic catgut. Gastro-hepatic omentum then closed around tube. Split tube and piece of gauze then sutured into gall-bladder fossa on under surface of liver, sutures passing through liver substance. Small glass tube to subhepatic fossa. Uneventful recovery.

Most of the internal fistulæ tend to close spontaneously, but the resulting adhesions often cause very distressing symptoms. These adhesions may cause pyloric obstruction, kinking of the common duct, interference with intestinal peristalsis, or even acute intestinal obstruction. If the internal biliary fistula remains patulous, and if the cystic duct is patulous, obstruction of the common duct may not produce any noteworthy symptoms, as the bile will be able to drain into the intestinal tract through the fistula. When the bile is discharged into the colon there are more

digestive symptoms than when it escapes into the duodenum or stomach. Interference with the function of the pancreas may occur, and may eventually demand operative relief. In a case reported by Leonardi the entire gall-bladder was discharged as a slough into the intestine through an internal biliary fistula, and was passed by rectum.

Treatment.—As the true lesion scarcely ever is recognized until after the abdomen has been opened, the surgeon must be prepared to meet the conditions as they exist. It is seldom possible and very rarely is it proper to return the various viscera involved in the adhesions to their normal relations. In most cases the intestinal or gastric opening can be closed by careful suture; if this narrows the lumen to a dangerous degree a short circuiting operation should supplement the repair of the fistulous opening. Only in rare cases is an intestinal resection required. The biliary side of the fistula is most easily relieved by cholecystectomy, when the fistula communicates with the gall-bladder or cystic duct; in such cases as well as in those where the common or hepatic duct are involved in the fistula, drainage of the main bile-channels (choledochus or hepaticus) should be instituted.

External Biliary Fistulæ.—Most of the external biliary fistulæ encountered at the present day are the result of an operation, and develop in the operative cicatrix. Those which form spontaneously, and which sometimes are called "pathological," may open almost at any point of the abdominal wall. The situation of the skin opening depends upon the size and position of the gall-bladder, and upon whether or not the fistula communicates directly with the gall-bladder or ducts, or passes through an intervening abscess cavity by a long and tortuous channel. In the series of cases collected by Courvoisier the fistulous openings were in the following situations:

In the right hypochondrium	40
At right costal border	36
On right side of epigastrium	17
In epigastrium	6
In right iliac fossa	10
Near umbilicus	22
At umbilicus	11
In left groin	1
Multiple openings	1
	153

External biliary fistulæ are classed as *complete* (*biliary*) or *incomplete* (*mucous*), according to whether or not they discharge bile. Practically all the spontaneous or so-called pathological fistulæ are complete. The diagnosis is made by the recognition of bile in the discharge. In most cases calculi also escape from time to time. In a case reported by Gutteridge a single stone 3 inches in diameter was discharged in this way. Usually such a fistula persists until all calculi present in the biliary passages have been discharged; it may then close of itself. But the period of time during which it will remain open cannot be foretold, and in most cases early operation is indicated to remove the remainng calculi, and restore the intestinal drainage of bile.

Abscess of Abdominal Wall from Perforation of Gall-bladder; Calculi in Gall-bladder. Cholecystostomy. Recovery.

M. R., female, aged forty-one years; admitted to German Hospital, October 20, 1904. Two brothers had had gall-stones. Patient never had had typhoid fever. Present illness began twenty-three years ago, with severe pains in epigastrium and vomiting; indigestion after eating, accompanied by much flatulence. Last attack of severe pain five years before present one, which has been more or less constant during last five months, with dull pain in gall-bladder region referred to back; lately pains have been sharp and shooting.

Examination.—Œdema of abdominal wall in gall-bladder region, with swelling, redness and tenderness. Marked tenderness in right hypochondrium. Hæmoglobin 61 per cent., W. B. C., 9200.

Operation, by Dr. Deaver. Ether anæsthesia. Incision over mass in abdominal wall which proved to be an abscess communicating with fistulous opening in the gall-bladder. Adhesions between gall-bladder, stomach, duodenum, and colon. Adhesions not disturbed. Liver enlarged and congested. Three small and one large calculi removed from gall-bladder through fistula. Drainage of gall-bladder. Recovery.

Postoperative fistulæ may be either mucous or biliary.

A postoperative mucous fistula is very rare if the gall-bladder has been removed; usually it indicates that the cystic duct is no longer patent, and that the gall-bladder is a useless appendage. The cystic duct usually is closed by cicatricial changes which are the result of previous disease, but in some instances closure is due to impaction of a calculus. There is a more or less constant flow of muco-purulent material from the fistula, but the discomfort produced may be very slight, the amount of the discharge seldom being

more than 30 to 40 c.c. (1 ounce) in twenty-four hours. Should the external opening close, however, the discharge will accumulate within the fistulous tract, causing severe pain and at times symptoms of septic absorption. Should operation be inadvisable for any reason, or should it be refused, the external opening of the fistula should be kept patulous by the use of a tube. The following case illustrates this condition, as well as the method of cure by operation.

Mary S., aged twenty-seven years, was operated upon in the German Hospital in 1908, for gall-stones, a cholecystostomy being performed. The sinus was almost closed when she went home. Shortly after it closed she had severe crampy pains in the region of operation. The fistula was opened, with immediate relief, and a rubber drainage tube was inserted into the tract. This was worn by the patient for over two years. The discharge consisted of white muco-purulent material. Two weeks before readmission to the German Hospital, the patient noticed a discharge of bile which continued profusely until the second operation.

Second operation, April 28, 1911. Ether anæsthesia. Incision made around old cicatrix, and the fistulous tract dissected down to the gall-bladder. The gall-bladder was removed, and all the ducts explored, with negative result. All the ducts were patent, and no calculi were present. Patient went home June 4, 1911, with the wound entirely healed.

In the *postoperative biliary fistula* a more serious condition is present. Usually there is an obstruction in the common duct, and bile is discharged continuously from the external opening of the fistula; as much as 1000 c.c. (one quart) of bile may be lost in twenty-four hours. If the discharge is not very profuse, the patient may be in very good health, but in most cases there is a certain amount of indigestion, and the patient complains of feeling "miserable." The discomfort caused by the discharge of bile usually is so great that operative measures should be instituted for its relief. But in a certain number of cases external drainage of bile after operation acts as a therapeutic measure in relieving the angeio-cholitis found at operation, and in permitting a subsidence of pancreatic lymphangeitis. In such cases it may be necessary to wait six to eight months or a year before it will be entirely safe to restore the bile to the intestinal tract; and before this time has elapsed the fistula may close spontaneously. So long as bacteriological examination shows the biliary discharge to be actively infected, we believe it is inadvisable to undertake

operation for its relief. Before operation is adopted even in cases where the discharge proves sterile, it is well to try irrigation of the tract. In this way it often is possible to dislodge calculi which may have been overlooked at the time of operation, or which may have descended from the intrahepatic bile-ducts since that time. The fluid used for irrigations may be olive oil, or a 0.5 per cent. solution of animal soap, as advised by Brockbank; or a solution of turpentine in ether, as recommended by Robson, especially when the obstruction is due to the presence of stones or fragments. A small soft catheter is passed down the fistula until an obstruction is encountered, when the irrigating fluid is forced gently into the catheter by the force of gravity, or from a syringe.

When operation is undertaken, the fistulous tract should be dissected out cautiously until the peritoneum is opened. Surrounding structures are then packed off, and the dissection continued until the biliary system is exposed. If an inoperable obstruction of the common duct is found, the surgeon will be very fortunate if a reasonably healthy gall-bladder has been left at the previous operation, since it may now be used to conduct the bile into the intestinal tract by means of a cholecystenterostomy. If the gall-bladder has already been removed, or if it requires removal at the second operation, some form of anastomosis will now have to be made between the common or hepatic duct above the obstruction, and the intestinal tract (page 124). If the obstruction of the common duct can be removed, this should of course be done; a calculus may be extracted or pushed into the duodenum; a stricture may be stretched or incised; a benign or even a malignant tumor may be extirpated. Where restoration of the discharge of bile through the common duct can be secured, the gall-bladder had best be removed. These operations never are easy, and may be very difficult.

Biliary Fistula. Cholecystectomy. Recovery.

John K., aged thirty-eight years, was admitted to the German Hospital November 25, 1911, with a history of having been operated upon for gall-stones three years previously. Biliary fistula persisted for eleven months after operation, the discharge of bile having been profuse for nine months. The fistula closed eleven months after operation and remained closed until

five weeks before admission to the German Hospital, when the wound opened after an attack of very severe abdominal pain. For one day the discharge was purulent and profuse; this was followed by a free discharge of bile. During the four days prior to admission the discharge had been entirely mucoid in character. There had been no jaundice since the original operation.

Operation, November 27, 1911. Ether anæsthesia. Scar dissected out. Dense adhesions found between the gall-bladder, duodenum, and omentum. Gall-bladder small and thickened. Cholecystectomy. No stones found in gall-bladder or duct. Patient made an uneventful recovery.

Biliary Fistula. Cholecystectomy; Choledochotomy. Death.

B. S., female, aged thirty-eight years, admitted to the German Hospital September 16, 1912, with a history of having been operated upon for gall-stones nine months previously. A biliary fistula persisted. There had been no attack of pain or jaundice. Bowels had been regular, but the stools had been light in color, soft, and of foul odor. The fistula discharged dark brown fluid mixed with mucus.

Operation, November 18, 1912. Ether anæsthesia. Scar dissected out. Adhesions found between the omentum, gall-bladder, under surface of the liver, and the abdominal wall. The liver was adherent to the parietal peritoneum and could not be displaced. Adhesions ligated and divided, and gall-bladder exposed. It was of good size, but its walls were markedly thickened, and a stone was encysted in its wall. Cholecystectomy. Large stone found in common duct. Choledochotomy, stone removed, and duct drained. Owing to the fixity of the liver it was impossible to ligate the cystic artery because of its depth, and two hæmostats were left in place. Drain placed in bed of gall-bladder and in the subhepatic space, and the wound in the abdominal wall partly closed. The patient did not react from the operation, and died twenty-four hours later.

REFERENCES.

Courvoisier: Path. u. Chir. d. Gallenwege, Leipzig 1890, S. 116.
Gutteridge: Lancet, 1878, i, 851.
Kelly: Osler's Modern Med., Phila., 1908, vol. v, p. 840.
Leonardi: Gaz. d. Osp . e. d. Clin., 1909, xxx, 322.

Intestinal Obstruction from Gall-stones.

Intestinal obstruction due to gall-stones is a complication or sequel of cholelithiasis that is not frequently seen, although it is not extremely rare. From statistics collected by Barnard,

Brockbank, Durham, Leichtenstern, and others, it is found that gall-stones are the cause of from 2 to 4 per cent. of cases of intestinal obstruction. About 250 cases are on record, according to Edwards. In a large majority of cases the calculus enters the intestinal tract through a fistulous opening between the gall-bladder and duodenum.

Porter suggests the following as a convenient classification of cases of intestinal obstruction from gall-stones: *Directly*, by plugging or corking the bowel (obturation): *Indirectly*, (1) by causing intestinal paresis; (2) by the production of volvulus; (3) by producing a stricture of the intestine; (4) by causing spasmodic contraction of the circular muscle fibres of the bowel; and (5) by producing angulation of the bowel.

The most frequent cause is *obturation*, or the impaction of the calculus in the intestine; the next most frequent causes are *volvulus* caused by violent peristaltic movements of the intestine in the effort to rid its lumen of the obstructing body; and *strictures* the result of ulceration caused by gall-stones which have been arrested for a time, but which may have been passed by rectum long before the stricture gives rise to symptoms. Occasionally gall-stones in their course through the intestinal tract may become lodged in *diverticula*. Dr. Henry Winsor has shown us photographs of the specimens from a remarkable case which came under his observation at autopsy, while in Manila, P. I. The small intestine presented innumerable diverticula, in which were lodged concretions presumably biliary in origin.

Obstruction from obturation by gall-stones may occur in any portion of the intestinal tract, from pylorus to anus. As the small intestine gradually narrows from its beginning to the ileo-cæcal valve, the position of the obstruction naturally varies with the size of the stone. Large calculi are arrested higher than smaller, and the latter may succeed in escaping from the body through the anus after causing slight or partial obstruction at various times. Very small calculi may be passed without producing symptoms of obstruction at any time. A very large calculus, measuring $4\frac{1}{2}$ by $3\frac{1}{2}$ inches in circumference was found impacted in the ileum in a case recorded by Roberts.

According to Courvoisier's statistics of fifty-two cases, the site of impaction was in the duodenum and jejunum in 21.4 per cent.;

in the ileum in 65.4 per cent.; at the ileocæcal valve in 10 per cent.; and in the sigmoid flexure in 2.4 per cent.

A case of pyloric obstruction due to gall-stone has been reported by LeBec and Müller: the patient complained of pain in the stomach accompanied by a marked burning sensation; there were signs of pyloric obstruction, and carcinoma of the pylorus was suspected. Laparotomy revealed a thickened pylorus, with strong adhesions to the gall-bladder, which contained stones. The gall-bladder was emptied, the adhesions released, and the pylorus excised. Instead of the suspected carcinoma, a gall-stone was found embedded in the thickened pyloric wall partially occluding its lumen. The patient did not survive.

The **symptoms** are those of intermittent obstruction, the obstructing body being more or less migratory. When obturation occurs, obstructive symptoms are noted at once. The initial symptoms may be very mild. Slight colicky pains, quickly subsiding, and leaving the patient apparently in perfect health, may occur for many hours before anything more serious is noted. Nausea appears early, and is followed by vomiting. Distention of the abdomen, which never is an early symptom, may never occur at all if the obstruction is very high in the intestinal tract. When the proximal portion of the bowel becomes very much distended, or reversed peristalsis is present the calculus may slip backward and relieve the obstruction. Under such circumstances the symptoms gradually subside and the patient returns to an apparently normal condition; but will again become the victim of obstruction when the concretion is forced down into a portion of the bowel with lumen too small to accommodate it.

In some cases the calculus can be felt through the abdominal walls as a round or slightly oblong hard mass, freely movable, and devoid of tenderness. The position of this mass may be in any portion of the abdomen or in the pelvis, in which latter position it may readily be mistaken for an ovarian neoplasm.

The **diagnosis** of gall-stone obstruction of the intestines cannot be made with certainty in most cases until the abdomen has been opened. A history of cholelithiasis, if obtainable, a history of previous similar attacks of obstruction, and the presence of a more or less movable hard mass, may lead to a recognition of the true condition.

The **treatment** is operative, except in a few cases where the symptoms of obstruction have been very mild and not of long duration. In such cases it may be advisable to temporize, trusting to Nature to expel the calculus. Under no circumstances should purges be given. If operation is not required, it is best to take measures to check peristalsis by administering nothing whatever by mouth, by applying ice to the abdomen, and even by giving morphine hypodermically in the hope that, intestinal spasm being relieved, the stone may pass onward without causing further trouble. If symptoms of obstruction are at all severe, however, operative treatment should be undertaken at once. Unless the obstruction can be accurately localized in another region, the abdominal incision should be made close to the mid-line, below the umbilicus. The hand is then introduced, the obstruction sought for, and the portion of intestine involved is brought out of the wound if possible, and well walled off with gauze. If the obstruction is not readily found time will be saved by evisceration. If the stone can be dislodged it should be pushed into a portion of the bowel that is free from the inflammatory changes caused by obstruction. If impacted in the lower ileum it may be possible to push it through the ileocæcal valve and to work it through the colon to the rectum, whence it can be removed by an assistant. If enterotomy is necessary to extract the stone, it should be held firmly against the wall of the intestine opposite the attachment of the mesentery, and a longitudinal incision made over the mass. This incision should be just large enough to allow expulsion of the calculus; and it should be made through healthy bowel, preferably on the aboral side of the obstruction. After removal of the stone the intestinal wound should be closed (1) with a continuous through-and-through suture of catgut, special attention being paid to inclusion of the mucous membrane in the suture, and to proper inversion of the wound margins; (2) this first row of sutures should then be reinforced with a continuous sero-serous suture (Lembert or Cushing) of fine linen. If it has been impossible to dislodge the calculus it may be necessary to resect the segment of bowel obstructed; this will also be necessary if gangrene has occurred from strangulation. In other cases the abdominal wound may be closed without drainage.

The high mortality of this condition is due to delay in resort-

ing to operation. F. Martin has collected nineteen operations done within recent years, including three of his own. Of these patients eight recovered, and eleven died, a mortality of 57.8 per cent. All three of Martin's own patients recovered, though in one resection was necessary.

REFERENCES.

Barnard: Annals of Surgery, 1902, xxxvi, 161.
Brockbank: quoted by Robson, loc. cit., p. 142.
Courvoisier: Path. u. Chir. d. Gallenwege, Leipzig, 1890, S. 101.
Durham: Quain's Dict. of Med., New York, 3d Ed., 1902, p. 774.
Edwards: Principles and Practice of Med., Phila., 1907, p. 674.
LeBec and Müller: Bull. et Mém. Soc. Anat. Paris, 1903, lxxviii, 72.
Leichtenstern: quoted by Robson, loc. cit., p. 142, p. 154.
Martin: Ann. of Surg., 1912, i, 725.
Moynihan: Med. Chronicle, 1903, v, 277.
Porter: Amer. J. of Obstet., 1906, liv, p. 693.
Robson: Dis. of Gall-bladder and Bile-ducts, London, 1904, 3d Ed., p. 153.
Roberts, W. O.: Louisville Monthly J. of Med. and Surg., 1902–03, ix, 114.

CHAPTER III.

SURGERY OF THE LIVER.

Anomalies of Size, Shape, and Position of the Liver.

These may be *congenital* or *acquired*. As the result of faulty development the liver may enter the thoracic cavity, may lie immediately beneath the skin in the region of the umbilicus (hepatomphalos), may form part of the contents of a congenital umbilical hernia; or, finally, in cases of transposition of the viscera, may be located on the left side of the body. In such cases the left lobe is larger than the right. Other congenital anomalies consist in absence of one or more of the ligaments; variation in the size of the lobes; or the presence of accessory lobes. In monsters the liver may be absent. Linguiform lobulation of the right lobe may be found as a congenital defect; such cases are mistaken at times for the acquired condition first described by Riedel (page 158). Lobulation or other change in the configuration of the entire organ may result from prenatal syphilis, tuberculosis, or hepatitis; though, according to Kelly, it is more likely that these conditions are acquired in postnatal life.

Acquired changes in the form of the liver are found frequently. It is a plastic organ, and whenever there is prolonged abnormal pressure or traction on the liver substance, lasting changes in form are produced. The principle cause of these acquired changes in the form of the organ is found in the pressure exerted by a corset or band. In the classical monograph of Hertz two principal forms of the so-called "corset-liver" are described: In the *first*, one or both lobes are elongated downward in the form of a thin apron, which may lie over or under the intestine; and the anterior surface of the liver is marked by a transverse depression (the "corset-furrow"), the overlying peritoneum being thickened and fibrous. In the *second* form the upper portion of the liver is much thicker than the lower. The posterior surface is curved around the spinal column while the anterior surface conforms to the concavity of the anterior abdominal wall. The transverse

corset-furrow is not so marked as in the first type. Mixed types may also be recognized.

The **"corset-liver"** is seen much more frequently in women than in men, although the latter are not free from it. The change in shape is due to pressure atrophy of the liver substance, followed by the formation of cicatricial tissue. The degree of alteration in shape, and the depth of the furrow vary considerably in different cases. In some instances continued pressure will cause atrophy

Fig. 16.—Corset Liver from a Patient in the Episcopal Hospital Who Died from Peritonitis Due to Perforation of a Carcinomatous Ulcer of the Stomach.

of practically all of the hepatic tissue in the furrow, nothing but a hinge of fibrous tissue remaining; while in others there will be a mere indentation on the convex surface of an otherwise normal liver.

The "corset-liver" is of interest to the surgeon mainly as an ætiological factor in diseases of the gall-bladder and bile-ducts. This aspect of the condition was discussed at page 88.

Linguiform lobulation of the liver (**Riedel's lobe**) is classified by Moynihan, Kehr, Kelly and others as a partial hepatoptosis; it was so graphically described by Riedel in 1888, that his name has become inseparably connected with the condition. It had been recognized previously and its true significance perceived by Terrier. The right lobe is the usual site of the deformity although in rare cases the quadrate, or even left lobe of the liver has been affected.

In the typical case there is no ptosis or increased mobility of the liver, but a simple elongation or hypertrophy of one section of the organ. The various structures holding the liver in place are intact, and the liver maintains its normal position against the diaphragm. On this account we believe the term "partial hepatoptosis" to be a misnomer.

Linguiform lobulation may result from tight lacing, being an extreme degree of "corset-liver"; but usually it is due to drag upon the liver by a distended gall-bladder, and in almost all cases is associated with disease of that viscus. According to Kelly enlargement of the gall-bladder has been found in 60 per cent. of all cases of lobulation.

A Riedel's lobe varies greatly in size and shape. There may be a scarcely appreciable elongation downward of the right lobe, or a freely movable, distinctly pedunculated "wandering lobe." In the latter instance the pedicle usually is attenuated, being little more than a fibrous cord which attaches the lobe to the liver. Under such conditions the lobe will be very freely movable. Riedel's lobe is not infrequently the seat of gummata, of abscess or tumor.

According to Harris, Leue in a total of 3484 autopsies found a constricted lobe in 1.9 per cent. of male subjects and in 25.3 per cent. of female subjects over sixteen years of age.

The *symptoms* of linguiform lobulation usually are those of an associated condition in the gall-bladder. There may be tenderness and pain, as a result of congestion of the anomalous lobe; or the lobe may exist for years without producing any symptoms. Distortion of the duodenum may cause dyspeptic symptoms.

Treatment.—This consists in proper treatment of any biliary lesion present. Cholecystostomy is the operation of choice; the liver usually returns to its normal shape after this operation. If no cause can be found to account for the development of the linguiform lobe, it may be excised, or sutured to the anterior abdominal wall.

Hepatoptosis, movable liver or **floating liver** may be *congenital* or *acquired.*

Congenital movable liver may be due to the presence of a mesohepar, as in the case reported by Clark and Dolley. Their patient was a female, thirty-four years of age, and unmarried. Her

abdomen was markedly asymmetric, there being a definite bulging of the right side from the costal margin to the iliac crest. The lower lobe had no ligaments, and there was a double reflection of the peritoneum from the upper lobe to the diaphragm, producing a true meso-hepar, which measured 13 mm. in length. Albu studied ninety-four infants ranging from one to ten days old, and found visceral ptosis in 11 per cent. of the males and 44 per cent. of the females; hepatoptosis was present in 5 per cent. of the males and in 9 per cent. of the females.

Acquired movable liver, which was first noted by Heister in 1754 in his study of a cadaver, and first described in the living subject by Cantani in 1866, is not a very rare condition. Clarke and Dolley have found reports of 118 cases; this includes those collected by Legg, Faure, Graham, and Ssaweljew. Of these, thirteen were in men and 103 in women, and one in a child. Of the women, ninety-three were married and ten single. In a study of 3400 patients, about equally divided as to sex, Albu found visceral ptosis in 21 per cent. of the men and 68 per cent. of the women; hepatoptosis was found in 9 per cent. of men and 17 per cent. of women.

The liver is naturally a mobile organ, moving upward and downward with the ordinary respiratory movements and also, to a slight degree, with posture. Normally, the liver is held in position by the inferior vena cava and the hepatic veins which empty into it; by the coronary ligaments and their cellulo-vascular bands; by the fibrous tissue found on the posterior extra-peritoneal surfaces; by the intra-abdominal pressure exerted by the muscles of the abdominal wall; by the so-called suspensory ligaments; and by negative intrathoracic pressure. Anything which decreases the sustaining powers of these various factors or markedly increases the amount of work thrown upon them may be a factor in causing displacement of the organ. The causes may be divided into the following classes: (1) traumatic; (2) conditions producing increased intra-abdominal capacity, such as repeated pregnancies when associated with pendulous abdomen, removal of tumors or ascitic fluid, etc.; (3) structural increase in the size of the liver, or solid growths dragging on the organ; and (4) the use of improperly fitting corsets. Downward traction may be exerted by an abnormally shortened round ligament. Cheyne believes that "once

the liver is displaced downward from the diaphragm, it is sucked down still more by the atmospheric pressure and the intestines pass up between it and the diaphragm." When Heister, in 1754, found a movable liver at autopsy, he advanced the theory that the development of the condition "was due to the will of God to prove his Omnipotent power."

Landau divides cases of hepatoptosis into three grades or degrees. In the *first* grade there is moderate descent combined with version, either anterior or posterior. In the *second*, there is marked descent combined with lateral displacement toward the right and version either anterior or posterior. In the *third* there is displacement directly downward, or slightly oblique, with the left lobe usually palpable in the abdominal cavity. The liver may also be displaced upward, with rotation, so that the inferior surface will present anteriorly.

The same factors which cause changes in the position of the liver frequently have a similar effect on the other organs of the abdominal cavity. In consequence of this a general *splanchnoptosis* usually is associated with movable liver. T. R. Brown quotes Glénard as having found, in a study of 1310 patients, fifty-one cases of hepatoptosis, thirty-two of which were associated with movable kidney.

Temporary displacement of the liver must not be confounded with movable liver, although the latter condition may be established ultimately. Such temporary displacement not infrequently is produced by pressure from pathological lesions distinct from the liver. Pressure may be exerted in a downward direction by intrathoracic conditions such as pleurisy with exudate, empyema, hydrothorax, or emphysema; by tumors or abcesses of the lungs or mediastinum; or by pathological changes between the diaphragm and the liver, such as subphrenic abscesses. Upward displacement may result from disease of the abdominal organs, especially when associated with ascites. A traumatic opening through the diaphragm may allow the liver to become involved in a diaphragmatic hernia. The resulting degree of mobility of the liver, after the subsidence of these various affections, will be modified by the extent and duration of the pressure exerted and by the strength and abundance of the resulting adhesions.

The *symptoms* of movable liver are rather vague. In many

cases the symptoms presented by the displaced liver are overshadowed by those of the diseases causing the trouble. In all of these cases the displaced liver is of secondary importance. In movable liver pain frequently will be the first symptom noticed. This pain may begin as the result of any jarring movement of the body, or anything causing a sudden spasmodic contraction of the diaphragm. The pain may be noticed first after jumping, sneezing, coughing, etc. Later, paroxysms of pain may occur without any known cause. The pain is most commonly felt in the right hypochondrium and epigastrium but it may radiate to the right shoulder or to the right flank. The pain is relieved almost immediately by replacement of the liver in its normal position. Pressure on the liver, when ptosed, causes peculiar sensations in various parts of the body, especially in the arms and shoulders. Digestive symptoms vary. Usually symptoms of gastric dyspepsia are present; especially flatulence, discomfort during the period of digestion, vague pains in the epigastrium and often throughout the intestinal tract, headache, insomnia, etc. There is a feeling of weight and heaviness in the epigastrium and hepatic region. Sometimes there may be found also ascites, polyuria, hemorrhoids, jaundice, recurring hemorrhages from the stomach and œdema of the lower limbs. A caput medusæ may be found as a result of traction on the vena cava. As a rule, there is no marked disturbance of the normal biliary activity, though gallstones often are present.

The *diagnosis* of hepatoptosis is based on the symptoms mentioned, together with a demonstration of the displacement. The proptosed liver will be recognized as a large tumor in the abdomen, of the size and consistency of the liver; usually it is to the right of the umbilicus, and often a distinct notch can be felt. The tumor may be partly replaced toward its normal position where it will remain while the patient is recumbent. When it is displaced, percussion of the normal site of the liver will give a tympanitic note rather than the usual dull or flat liver note, and the pulmonary resonance posteriorly will merge with intestinal resonance. It may be distinguished from a movable or enlarged kidney by the absence of urinary symptoms; by observing that the liver moves during respiration; that it lies in front of the colon, not behind it; and by attention to the changes in the percussion note in the nor-

mal hepatic area, to which attention already has been directed. Fluoroscopic examination should be made or an *x*-ray photograph should be obtained for confirmatory evidence.

The *treatment* is palliative or operative. *Palliative treatment* consists in the application of a well-fitting binder or abdominal bandage which will retain the organ in its normal site after it has been replaced by manipulation. The pressure should always be from below upward, the intestines being pushed upward to act as a cushion on which the liver may rest. Moynihan thinks the inflatable pad of Byron Robinson the best apparatus for this purpose.

Operative treatment consists in retaining the liver in its normal position by one of the numerous methods of hepatopexy that have been devised. In replacing the liver the organ should be rotated backward and to the right, and pushed well up against the diaphragm, after examination of the space between the liver and diaphragm has shown that no coil of intestine lies between.

The first operation of hepatopexy was performed by Gérard-Marchant in 1891. He sutured the anterior edge of the liver to the costal margin with silk. Péan held the liver in its normal site by first making a transverse abdominal incision, replacing the liver, and then making a barrier below it by suturing the anterior and posterior layers of parietal peritoneum together transversely. Franke united the anterior margin of the liver, with the exception of the portion near the gall-bladder, to the costal margin and then placed gauze between the upper surface of the liver and the diaphragm. The gauze was allowed to remain in place for eight days. Its removal was followed by the formation of strong adhesions between the liver and diaphragm. Moynihan advocates the same line of procedure, with the addition of gauze packing placed below the right lobe of the liver. Cheyne sponges the upper surface of the liver with undiluted carbolic acid. He also recommends division of the round ligament when that structure seems too short. In all forms of operation the patient must remain on the back in bed for at least four weeks, at absolute rest. Elevation of the foot of the bed a few inches will also aid in overcoming any tendency of the liver to prolapse during the formation of strong adhesions. For some months after operation a

well-fitting abdominal belt or binder should be worn, and all violent efforts should be carefully avoided.

REFERENCES.

Albu: Berl. klin. Woch., 1909, xlvi, 289.
Brown, T. R.: Osler's Modern Med., Phila. and N. Y., 1908, v, 606.
Cantani: Annali Univ. di Med. e Chir., Milano, 1866, cxcviii, 373.
Cheyne: Lancet, 1906, i, 947.
Clarke and Dolley: Amer. Jour. Med. Sc., 1905, ii, 969.
Gérard-Marchant: Gaz. d. Hôpitaux, 1891, lxiv, 878.
Harris: Jour. Amer. Med. Assoc., 1910, i, 397.
Heister: Acta Phys.-Med. Nat. Curios., Norimbergæ, 1754, x, 1.
Hertz: Abn. in d. Lage u. Form d. Bauchorgane bei dem erwachsenen Weibe eine Folge des Schnürens u. Hängebauches. Berlin, 1894.
Kehr: System of Practical Surgery, Ed. by v. Bergmann, Bull, et al., Phila. and N. Y., 1904, vol. iv, p. 631.
Kelly, A. O. J.: Osler's Modern Medicine, Phila. and New York, 1908, vol. v, p. 699.
Landau: Die Wanderleber u. der Hängebauch der Frauen, Berlin, 1885.
Leue: Trans. West. Surg. and Gynec. Assoc., 1910, (Quoted by Harris, loc. cit.)
Moynihan: Abdominal Operations, 2nd Ed., Phila., 1906, p. 138.
Riedel: Berl. klin. Woch., 1888, xxv, 577; 602.
Robinson, Byron: Med. Age, Detroit, 1903, xxi, 405.
Terrier and Baudouin: Progres. Mèd., Paris, 1888, 2e. S., viii, 121.

Abscess of the Liver

Abscess of the liver (**suppurative hepatitis**) may be classified in various ways: as *single* or *multiple;* as *tropical* (amœbic) or *non-tropical;* as *primary* (traumatic) or *secondary;* according to the *character of the infecting micro-organism;* or according to its *location*, the "anatomical" classification. Kelly gives Cantlie's anatomical classification as follows: "(1) Suprahepatic abscess, a collection of pus between the layers of the broad (coronary) ligament of the liver, bounded above by the diaphragm and below by the liver where it is destitute of peritoneal covering. This form of abscess is due to inflammation of the lymphatic vessels of the upper surface of the liver and is independent of dysentery and other intestinal lesions. (2) Intrahepatic abscess, a collection of pus within the liver substance, which comprises the great majority of cases. (3) Subhepatic abscess, a collection of pus

beneath the under surface of the liver and peritoneum, pointing in the epigastrium and due to lymphangeitis in the subhepatic region."

Probably the best classification is that which recognizes 1. **Traumatic abscess,** in which there is a wound or contusion of the liver substance followed by infection from the exterior or from the blood. 2. **Pyæmic (embolic) abscess,** in which infection reaches the liver by means of infected emboli, through extension of suppurative processes adjacent to the liver, by direct infection, etc. 3. **Amœbic (tropical) abscess,** in which the amœba coli is the chief ætiological factor. Rarer forms of abscess in which the liver becomes honey-combed with cavities containing pus and varying in size from a pea to a walnut, sometimes larger, are due to **tuberculosis,** or **actinomycosis.** Suppuration also frequently occurs in an **hydatid cyst** (page 189).

Ætiology. Predisposing Causes.—*Traumatic abscess*, as its name implies always is preceded by a wound or contusion involving the integrity of the liver cells. In cases of penetrating wound, by stab or gunshot injury, or when the liver is punctured by the fragment of a rib in compound fracture, the infection is admitted from without. In cases of contusion, or subscapsular rupture of the liver, the hæmatoma which forms may be converted into an abscess by infection through the blood, or lymphatics, or even through the biliary tract (page 170). *Pyæmic abscess* may have as a predisposing cause any suppurative, infectious, or parasitic condition in any portion of the body. Especially frequent are lesions in the distribution of the portal vein, such as **appendicitis** or **typhoid fever.**

Melchior gives the following figures showing the frequency with which typhoid fever is complicated by hepatic abscess.

Author.	Cases of Liver Abscess.	Cases of Typhoid Fever.	Percentage.
Holscher	12	2000	0.6
Horton-Smith	2	....	0.58
Piorkowsky	2	1229	0.16
Vierhuffs	3	1186	0.25
Berg	0	1662	....

The hepatic abscess usually develops during convalescence from typhoid fever, but a year has elapsed in some cases. The

average fever-free interval is fourteen days—the period between defervescence from typhoid fever and the development of symptoms from the hepatic abscess. The infection may reach the liver through the bile-ducts, through the systemic circulation, or by way of the portal vein. Four cases, all fatal, followed appendicitis of typhoidal origin. Melchior collects records of twenty-five cases, in nine of which no operation was done. Of these nine patients only two recovered, the abscess in the first case (1869) discharging spontaneously through the lung, and that of the second patient (1875) rupturing through the bowels. In four cases no formal operation was done, the abscess merely being punctured; all of these patients died. Of twelve patients in whom the abscess was treated by incision, in the ordinary way, only two died, a mortality of 17 per cent. This clearly demonstrates that the proper treatment of hepatic abscess in typhoid fever is by operation, according to the usual technique (Chapter IX).

As regards *hepatic abscess of appendicular origin*, it usually has been taught that the foci of suppuration in the liver are multiple and widespread; and there is no doubt that this is so in most cases, and that the condition is exceedingly fatal. Personally we never have seen recovery occur in a case of hepatic abscess secondary to appendicitis; but such cases have been reported in a few instances. Quénu and Mathieu, in their recent admirable study of this question, note that although foci of suppuration in almost all these cases are multiple, yet that some patients have had only one or at most two solitary abscesses, and that in other cases the abscesses, even if multiple, are closely congregated in the right lobe of the liver, and should be amenable to operative treatment. Loison, in 1900, reported twelve such cases, all the patients having died without operation because the possibility of cure by operation was not recognized. Loison reported one patient under his own care, whose life was saved by timely operation, and he says that Körte in 1892 also had one patient who recovered after operation. We are inclined, however, to agree with Tuffier, who in the discussion which followed the reading of Loison's report said (l. c., page 669) that to him the diagnosis in Loison's patient seemed uncertain; it might have been a case of subphrenic abscess following appendicitis. Quénu and Mathieu collected records of fourteen operations for this complication of appendicitis, with only

two deaths. The cases suitable for surgical intervention are not those presenting the usual picture of diffuse suppurative hepatitis or pylephlebitis closely following the attack of appendicitis, but those in which the symptoms of liver abscess develop after a free interval, and in which the physical signs indicate that a single abscess may be present. The operative methods in these cases are the same as in cases of tropical abscess of the liver (Chapter IX).

Abscesses as a result of suppurative processes in the structures adjacent to the liver are not common. They may follow acute empyema of the gall-bladder, perforation occurring through the portion of that viscus which is in contact with the liver substance, involvement of the liver and subsequent abscess formation resulting (Case history, D. E., page 73). Direct extension from diseased conditions of the pylorus and pancreas, after the formation of adhesions binding these structures to the liver, may result in infection and abscess formation in the liver; but this is rare. Liver abscess due to *suppurative cholangeitis* almost invariably is multiple and a sequel of lesions of the biliary apparatus. Theoretically it is possible for such intestinal affections as typhoid fever, gastroduodenitis, duodenal ulcer, etc., to infect the bile-ducts by continuity of structure, but in almost all such cases it is quite evident that the foci of suppuration in the liver are due to septic emboli received through the portal system or by way of the systemic circulation, as in pyæmia.

Liver abscess has been known to follow *influenza, yellow fever* and many other *infectious diseases*. Suppurative venous thrombosis with the production of septic emboli and abscess of the liver may follow almost any infective process in the body either by way of the portal or the systemic circulation. These predisposing causes have been well summarized by Munro, as follows: "Malaria, infections of the thoracic organs or of the umbilicus, pyæmia arising from various infections of any portion of the body, anthrax, ulcers of the intestinal tract, pelvic infections, splenic abscess, abscess of the mesenteric lymph-nodes, infections of the biliary tract and pancreas, from peritonitis, echinococcus, infection after hæmorrhoidal operations, etc."

McWilliams in an analysis of sixteen cases of abscess of the liver found that ten of them exhibited no apparent or evident ætiological factor.

Climate—Climate plays a very important part as a predisposing cause of amœbic, or tropical, abscess. It is a change of climate, however, rather than the climate itself which is the important factor. Tropical dysentery, which is so frequently the forerunner of abscess of the liver, occurs most frequently among those who go to the tropics from a temperate climate; and especially among those who do not conform to the diet and customs of the tropics. Herrick, from a study of abscess of the liver in the Panama Canal Zone, estimates that the white man from the United States is the most susceptible of all laborers, and that the colored employees from the islands are the least susceptible. He found one case in every 1178 whites from the United States; one case in every 2240 whites from Europe; and one case in every 3722 colored employees. Kieffer studied thirty-three cases of liver abscess among the soldiers in the Philippines, and found a history of dysentery in each; among twenty-five cases of abscess among the natives and civilians there was a history of dysentery in twenty-two. Kieffer concluded from his investigations that 20 to 25 per cent. of the cases of severe amœbic dysentery result in liver abscess; and that 85 per cent. of tropical abscesses are due to the amœba.

The frequency of tropical abscess as a result of dysentery varies with the statistics of different countries. In India, about 35 per cent. of the soldiers who died from dysentery had pus in the liver, while Rhoads found liver abscess in less than 5 per cent. of the patients in the Philippines who suffered from dysentery. Rhoads believes that all cases of amœbic abscess of the liver have been preceded by a dysenteric ulceration of the intestine. In those cases where causal relation cannot be determined, Rhoads believes the symptoms of dysentery were so slight that the patient forgot all about them, or that the ulcerations were slight and that the condition ran a latent course without the production of marked symptoms.

Coffin found 1523 cases of dysentery among 10,603 patients treated in the United States Army Division Hospital in Manila in three and one-half years. Of these, 859 were of the amœbic type; 236 were of the catarrhal type; and in 428 cases the type was not noted. The majority of cases were sent home invalided, in

from one to eight weeks, this fact accounting for the small number of liver abscesses found, thirty-four.

The disease is particularly prevalent in the Philippine Islands, India, and Egypt.

Exciting Causes.—All abscesses of the liver are the result of bacterial or parasitic invasion. Foreign bodies, such as fish-bones, pieces of straw, etc., have been found in the contents of the abscess cavity, but it is probable that the abscess resulted from the presence of micro-organisms and was not caused by the foreign body alone. Coccidia and the ray fungus of actinomycosis have been underlying factors; in rare instances the bacillus of tuberculosis may act as the direct causative factor.

Bacteria may gain entrance to the liver through a wound communicating with the exterior of the body; through extension of suppurating conditions in the adjacent structures; through the portal vein, the hepatic artery, the hepatic vein, or the lymphatics from adjacent or far distant points of infection; or through the biliary passages. The most frequent pathway of entrance is through the portal vein, the radicals of which carry the infection from more or less distant foci. In other than tropical climes the most frequent source of infection is found in disease of the appendix; in tropical countries the infection most frequently is a sequel of dysentery.

The micro-organisms which are found most frequently in liver abscesses are the Amœba coli, the B. coli, the B. pyocyaneus, the B. dysenteriæ, the D. pneumoniæ, the Actinomyces bovi, the S. pyogenes aureus and albus, and the B. typhosus.

Pathology.—Liver abscess may be solitary or multiple. Traumatic abscesses usually are single; pyæmic (embolic) abscesses usually are multiple; amœbic (tropical) abscesses usually are single; tuberculous abscesses usually are multiple. Zancarol found multiple abscesses in 38 per cent. of 211 cases; Jimenez, in 6 per cent. of 297 cases. The majority of abscesses develop in the right lobe of the liver, and usually are nearer the convex than the concave surface. They vary greatly in size, from minute foci to collections of pus that may occupy the greater part of the entire organ.

The pathological changes found in the liver vary with the underlying ætiological factors causing the abscess. The contents

of the abscess may vary in color from yellow to green or brown; in almost all cases microscopical study reveals the presence of bacteria in addition to liver cells and detritus, which are always present. The bacteria usually found are streptococci, staphylococci and colon bacilli. The walls of the abscess cavity are irregular and are composed of infiltrated liver substance.

Traumatic abscess always is septic. When there is a subparietal injury, the bacteria of suppuration may be introduced into the devitalized portion of the liver either through the bile-channels, the portal vein, the hepatic artery or the hepatic veins, or directly from the liver substance where there has been previous disease of the organ. When there is an external communication with the liver the germs of suppuration usually are introduced through this channel, frequently as a result of unnecessary probing and examination of the wound. The site of an abscess following trauma is determined by the site of the injury. The size varies with the extent of the injury and the severity of the infecting micro-organism.

In embolic or pyæmic abscesses, which comprise those usually seen in temperate climes, the process is somewhat different from that found in the traumatic abscess. Emboli carrying germs, lodge in one or more of the terminations of the portal vein, with resulting congestion, liquefaction and purulent degeneration of the liver substance. In many instances the emboli may be carried to the liver through the hepatic artery, the emboli passing through the capillaries of the lung, entering the left side of the heart, and then being carried with the arterial stream to the liver or other organs where abscesses are formed. It is probable that actual emboli do not pass through the pulmonary capillaries, but that clumps of bacteria are carried into the systemic circulation, and that these, when they are arrested in the next set of capillaries, there induce thrombosis and suppuration.

Pyæmic (embolic) abscesses usually are multiple and may occupy any portion of the liver substance, although they are found most frequently in the right lobe. They generally are near the surface of the liver and vary in size from minute foci of suppuration to masses the size of an entire lobe. Small foci frequently coalesce to form large abscesses. Microscopical examination of the contents of embolic abscesses shows the presence of active

bacteria in all instances. The liver substance surrounding the suppurating process always shows inflammatory changes.

In the *tropical* or *amœbic liver abscess*, the destructive process begins in the hepatic cells, according to Ewald. This process sometimes begins immediately after the onset of amœbic dysentery; but in most cases weeks, months, or even years elapse before the destructive process is manifest. Musgrave and Clegg have established the fact that amœbæ are amenable to changes in environment and that the power to propagate in temperate climes depends on the similarity to the old environment, and possibly on their ability to produce lesions in the tissues. The development of the abscess may be very slow.

Purulent softening causes coalescence of adjacent acini until the destructive process may involve an entire lobe of the liver. The abscesses are solitary in about 60 per cent. of the cases, and are most frequently found in the right lobe. Among 240 cases studied by Waring, the right lobe was affected in 163. Rouis, in an analysis of 156 cases, found the abscess in the right lobe in 122; and Musgrave and Clegg found the right lobe involved in 95 per cent. of their cases. Langenbuch explains this preference for the right lobe by the distribution of the branches of the portal vein. The branch going to the right lobe is larger than that to the left, and runs parallel to the course of the vein, while that to the left lobe runs at right angles. The separate currents in the portal vein were alluded to in Vol. I (page 45); the fact that most of the usual foci of infection are in the area which drains into the right lobe is another reason for the frequency with which this portion of the liver is affected.

When the abscess is within the right lobe the liver may be normal on inspection even if the abscess is large. Distinct elevation will be noticed as the abscess nears the surface. Peritoneal irritation usually is followed by the formation of adhesions between the liver and the abdominal wall, the adjacent viscera, or the diaphragm. In many cases some evidences of pleuritis are found.

Amœbic abscesses vary in size from minute foci of pus to a collection the size of the liver itself; almost the entire liver may be converted into a sac, and as much as 8000 c.c. have been removed from such a sac.

The contents of the amœbic abscess vary with the duration of the infection and with the extent of the destructive process. The consistency varies from fluid to gelatinous. The color usually is brown. As a rule, bile is not a constituent of the abscess, the contents being composed almost exclusively of liver cells and detritus. In cases of very long duration, yellow pus may be found.

Microscopical examination usually reveals amœbæ, especially in specimens obtained from the walls of the abscess. Bacteria are not present as a rule. Kieffer was able to demonstrate neither bacteria nor amœbæ in 20 per cent. of his cases; in 60 per cent. bacteria were present at the time of his examination. The amœbæ are demonstrable in the majority of cases, although it may be necessary to wait until there has been a discharge from the wound for three or four days before they can be found.

The wall of the abscess consists of liver substance which is soft and irregular and exhibits masses of necrotic liver tissue. Surrounding the abscess wall may be found areas of necrosis which usually do not present signs of suppuration.

Symptomatology.—The symptoms of abscess of the liver vary with the nature and severity of the preexisting condition, with the pathway of infection, and with the nature and virulence of the infecting micro-organism. In a great many instances, the symptoms are overshadowed by those of a coexisting inflammatory disease, especially of the biliary tract. In other instances the abscess is latent, giving rise to no symptoms, and being found unexpectedly at autopsy.

In *traumatic abscess* there usually is pain in the liver region which is markedly increased by pressure. Change of position will often cause increase in pain. If the abscess be located in the upper part of the right lobe pain will often be referred to the shoulder, a small branch of the phrenic nerve conveying the impulse of irritation to the nerve to the subclavius muscle (Bradshaw). A palpable friction rub may be elicited in some cases where perihepatitis is present. If there is involvement of, or pressure upon, the biliary ducts, jaundice will be present. The liver usually is enlarged, more or less irregular in outline, and a fluctuating mass possibly may be detected. Chills, fever, sweating, and a high leukocytosis generally are present. If an external

wound allows the discharge of pus, hepatic cells will be found in the pus or in the scrapings.

In *pyæmic* (*embolic*) *abscess* the symptoms may be entirely overshadowed, as pointed out by Kelly, by the symptoms of the primary disorder. As a rule the hepatic symptoms, if any are noticed, develop while the primary affection, such as appendicitis, is still active. When the liver complication remains latent for a time, as sometimes is the case according to Quénu and Mathieu when the infection follows acute appendicitis, the symptoms may not become evident until weeks after the entire disappearance of all appendiceal symptoms (page 166). In these cases the general condition of the patient may not be satisfactory and possibly there may be a persistent pallor with emaciation, but a comparatively long free interval will elapse before the symptoms of acute suppurative hepatitis assert themselves. In such cases the course resembles that seen in amœbic abscess. In the usual type of case, the most significant local symptoms are *pain* in the hepatic region, often referred to the right shoulder, and *tenderness* of the liver with *enlargement* of that organ. The general symptoms consist of *chills*, *sweats*, *fever* of the remittent type, and a marked *increase in the leukocytes*.

The following case well illustrates the difficulties of diagnosis, the exceedingly fatal prognosis, and the uncertainty as to the pathogenesis in many cases of diffuse suppurative hepatitis.

B. H., female, aged fifty-four, admitted to the German Hospital February 20, 1912, complaining of pain in the upper right side of the abdomen. Family history negative. Had typhoid fever twelve years ago. *Present illness* began three weeks prior to admission when she became chilled from exposure and was confined to bed for a few days. Then noticed fullness of upper abdomen and had great eructations of gas. This was followed by severe pain in upper right quadrant of the abdomen which lasted half the night and left the abdominal muscles very sore and stiff. First noticed jaundice one week prior to admission. The attack of pain was accompanied and followed by nausea and vomiting, high-colored, scanty urine, and "clay-colored" stools.

On admission patient seemed profoundly septic. Temperature was 102.6°, pulse 110, and respiration thirty-two. The abdominal muscles were rigid, especially marked in the upper right quadrant, marked tenderness above and to the right side of the umbilicus, with greatest tenderness along the costal margins. Liver dullness extended below the costal margin. Lower abdomen negative.

Blood Examination, February 21, 1912.—Hb., 66 per cent.; R. B. C., 3,930,000; W. B. C., 25,000; Polys., 94.5 per cent; Lymph., 5.5 per cent. Coagulation time, twelve minutes.

February 24, 1912: Hb., 60 per cent.; R. B. C., 3,390,000; W. B. C., 33,600; Polys., 93 per cent., Lymph., 6.5 per cent., Trans., 5 per cent. Coagulation time fifteen minutes.

February 26, 1912: Hb., 52 per cent.; R. B. C., 3,180,000; W. B. C. 70,700; Polys. 87.5 per cent., Lymph., 12.5 per cent.

Day after admission the liver seemed to be enlarged and very tender. Lymphangeitis marked. Patient grew progressively worse and died six days after admission.

Autopsy.—Gall-bladder thickened and inflamed, containing gall-stones and pus. The cystic duct was thickened and obstructed. There was stenosis of the common duct. The lymphatic nodes were enlarged. The liver was riddled with small foci of pus. Duodenum, stomach and other abdominal viscera negative, except for a few adhesions between the gall-bladder and the duodenum. It remains uncertain whether this case should be classed as one of suppurative cholangeitis or pylephlebitis.

Through the courtesy of Dr. Homer C. Bloom, with whom I was associated, I am able to report the following case:

E. B., fifty-three years of age; native of France; had had frequent attacks of what he said his doctors called gall-stone colic; these attacks dated back ten years; had been jaundiced more or less ever since the first attack; drank rather heavily of alcoholic stimulants; was a large eater. Had been losing strength and flesh rapidly during the past year, more or less gastro-intestinal disturbance, marked constipation.

April 22, 1905, patient suffering severe and agonizing pain over the region of the gall-bladder; there was marked tenderness over this region as well as over the upper part of the abdomen and liver. Jaundice almost bronze in color. The attack had been ushered in by a chill. There was marked dullness over the entire right side, extending three inches below the lower rib and as high as the right nipple and posteriorly to the level of the angle of the scapula. There was considerable distention of the abdomen and pronounced rigidity over the region of the gall-bladder.

Attacks continued at longer and shorter intervals until May 8, when operation was performed.

Operation.—Common duct distended with small stones and débris; common duct opened and emptied, but immediately from the liver the same character of material escaped in large amounts. A drainage tube was put in the common duct and the same material with bile continued to drain until the death of the patient.

Postmortem revealed a tremendous liver at least three times the weight of a normal liver; all through the structure of the liver were minute abscesses, and in that part nearest the ducts were hundreds of biliary stones

the size of a small pea. The kidneys were diseased, the right side of the heart dilated and the abdominal cavity filled with bloody fluid. A section of the liver is shown in the Frontispiece to this volume.

In *amœbic (tropical) abscess* the symptoms, in 33 per cent. of the cases, are entirely latent, the abscess either remaining quiescent or growing so insidiously that rupture gives rise to the first demonstrable symptoms (Rouis). Spontaneous rupture is a common event in large abscesses, Cyr finding a rupture in 159 cases in a series of 563, or in about 28 per cent. The abscess opened into the lungs in fifty-nine, into the pleura in thirty-one, into the pericardium in one, into the peritoneum in thirty-nine, into the intestine in thirteen, into the stomach in eight, into the kidney in two cases. In the great majority of all cases (nearly 60 per cent.) rupture occurs through the diaphragm. Flexner has reported two instances of rupture into the inferior vena cava.

The onset of the amœbic abscess usually is slow. The patient for months may complain of general ill-health, malaise and increasing weakness, without evincing any symptoms that refer directly to the liver. When there is a previous history of life in the tropics, of dysentery or other marked intestinal disturbance, these general symptoms become significant and a careful examination of the hepatic region and of the stools should be made. Pain is entirely absent in about 20 per cent. of cases. In other cases there will be a dragging sensation and some discomfort. When the abscess approaches the peritoneum, there will be actual pain, at times sharp and stabbing. The pain seldom is referred to the shoulder, according to Rhoads, unless the abscess is near the surface, near the gall-bladder, or in the Spigelian lobe. The temperature may be normal or of a severe septic type, depending upon the presence or absence of pyogenic bacteria in the abscess. In the purely amœbic cases, there is very slight increase of leukocytes with a corresponding increase of polynuclears. Jaundice will not be present unless there is pressure on the larger branches of the hepatic ducts.

Diagnosis.—In typical cases a diagnosis usually is not difficult. In other cases, either because the symptoms vary so greatly from the type, or because they are absent, a diagnosis may be very difficult to make. A provisional diagnosis should be made from the history of the case, the general appearance of the patient,

and the clinical picture presented. If the patient's physical condition is not very serious, further study generally will make the diagnosis clear. Exploration of the liver with a trocar never should be practised on account of the great danger of infecting either the peritoneal or the pleural cavity. If exploration is necessary to establish a diagnosis, an incision should be made of sufficient size to expose the liver and permit its examination by the fingers and the exploring needle under full control of vision.

Skiagraphic examination is of value in showing the extent of the swelling of the liver, and in determining its upper border. It is of little value in arriving at a differential diagnosis, but may be of great aid in determining the size, position, and mobility of the liver.

In *traumatic abscess*, where a communication leads from the exterior to the abscess cavity, the diagnosis may be cleared up at once by finding liver cells in the pus being discharged. Where there is no external wound, the diagnosis depends on the history of traumatism followed by symptoms which point to a lesion of the liver accompanied by fever of a septic type, chills, etc.

In *pyæmic* (*embolic*) *abscesses* the diagnosis cannot be made readily if the symptoms of the primary disorder are at all prominent. The general symptoms of involvement of the liver during, or following, any acute infectious disease or any suppurative process should make the diagnosis of abscess probable. Usually there will be fever, chills, sweats, marked increase of leukocytes, and sometimes jaundice, enlargement of the liver, with pain and tenderness beneath the right costal margins (see case history, page 173).

A mistaken diagnosis may be made, as is shown in the following cases.

A. M., male, aged forty-two years, admitted to the German Hospital December 3, 1912. In 1893 the patient had been in the British Army in Egypt and had had a severe attack of dysentery. During the four years he remained in Egypt he had had repeated attacks of dysentery and lost considerable weight. Since leaving Egypt, his bowels had been regular, but were moderately loose at times although there was no return of the dysentery.

Five months prior to admission to the hospital, he was kicked in the abdomen by a horse and was in bed from the effects of the injury for five weeks. He then returned to work but had to stop and return to bed. Vomiting began soon after the reception of the injury and has been more or less

persistent ever since. He has lost ten pounds in weight. Has not been jaundiced at any time. Two days before admission, patient had a severe chill which lasted for two hours.

On admission, examination revealed a mass the size of a grape fruit centering in the gall-bladder region, somewhat nodular and evidently part of the liver. It extended below the ribs in the mid-costal line. The mass was tender. Abdomen otherwise negative. Diagnosis of liver abscess made.

Operation, December 7, 1912.—Ether anæsthesia. Incision through right rectus muscle. Examination of the mass proved it to be located entirely within the gastro-hepatic omentum, the liver being apparently normal. A trocar and canula introduced into the mass and a considerable quantity of dark fluid aspirated. No rupture of the common duct detected. Cavity drained and wound closed to drainage. Examination of the aspirated fluid showed it to be blood, and free from bile. Patient recovered, and left the hospital January 13, 1913.

Mrs. O., aged 55 years, admitted to the German Hospital February 24, 1902. Family history negative. Has always enjoyed good health, except for asthmatic attacks during past few years. Has a large, healthy family.

Twenty-seven days before admission complained of languid feeling, constipation, and very slight jaundice. Later the patent developed fever of irregular type with chills. Following the chill she would sweat profusely and become very much exhausted.

On admission patient presented slight general jaundice, the skin being hot and dry. Lungs were emphysematous, respirations rapid, râles detected over both lungs. Abdomen was large. Liver extended one inch below costal margin; gall-bladder enlarged and exquisitely tender to palpation. Abdomen otherwise negative.

Temperature on admission 103.2° F., Hb. 64 per cent.; R. B. C., 3,100,000; W. B. C., 26,600.

Diagnosis of acute suppurative cholecystitis was made, and operation advised.

Operation, February 26, 1902.—Ether anæsthesia. Incision through right rectus. Peritoneum opened and gall-bladder exposed. Intestine walled off with gauze, and examination made of bile-ducts. All found to be normal. Gall-bladder was slightly enlarged, bluish in color and freely movable. No signs of inflammation outside the gall-bladder. Liver seemed harder than normal, but of normal color. No bulging of surface, no signs of inflammation, no adhesions. Gall-bladder sutured to parietal peritoneum without opening it. Wound closed to packing over gall-bladder with through-and-through silkworm-gut sutures. Patient left operating-table in fair condition, although she had taken her ether badly.

February 27, 1902: Temperature of patient 104.4° F. Packing removed from wound and gall-bladder opened allowing discharge of bile, but no pus. Drainage tube inserted into gall-bladder. Bowels moving freely.

Chills, high fever, and sweats continued, patient became gradually worse and died on the sixth day after operation.

Postmortem examination showed multiple abscesses of the liver and multiple foci of suppuration in both lungs.

In *amœbic (tropical) abscess*, the diagnosis often is attended by much difficulty. Here the past history is most important. With a history of dysentery or a sojourn in tropical climes and the presence of pain in the region of the liver, often referred to the right shoulder, tenderness and progressive enlargement of the liver, leukocytosis, especially if increased in the evening, fever, chills and sweating, the diagnosis of amœbic abscess may be made. The fever will vary from a very mild to a moderate or a severe degree, depending upon the presence or absence of pus-forming micro-organisms. When the amœbic abscess becomes secondarily infected with pyogenic bacteria the symptoms will be much more severe than in the simple amœbic cases. The leukocytes will vary from 15,000 to 30,000, part of which increase may be due to the intestinal disease. According to McDill, colonic irrigations and saline purgatives will eliminate the leukocytosis due to the intestinal infection. One of the best indicators of the presence of pus is a high leukocyte count in the afternoon. Examination of ordinary stools may be negative. McDill claims, however, that if amœbæ are present they always will be found in the third or fourth watery stool following the administration of saline purges. It must be remembered that the amœbic abscess may be latent and may remain so for an indefinite period. Manson states that "the most common mistakes in diagnosis are: (1) Failure to recognize the presence of disease of any description, even when an enormous abscess may occupy the liver. (2) Misinterpretation of the significance and nature of a basic pneumonia, a condition so often accompanying suppurative hepatitis. (3) Attributing the fever symptomatic of liver abscess to malaria. (4) Mistaking other diseases for abscess of the liver and *vice versa*—for example, hepatitis of a non-suppurative nature, such as that attending malarial attacks: suppurative hepatitis before the formation of abscess; syphilitic disease of the liver—softening gummata which are often attended with fever of hectic type; pylephlebitis; suppurating hydatid; gall-stone and inflammation of the gall-bladder; subphrenic abscess; abscess of the abdominal or thoracic wall; pleurisy; encysted empyema; pyelitis of the right kidney; pernicious anæmia; leukocythæmia; scurvy and

other similar blood diseases associated with enlargement of the liver; ulcerative endocarditis; kala-azar; Malta fever; trypanosomiasis. Any of these may be attended with fever of hectic type, increased area of hepatic percussion dullness, and pain in and about the liver."

In malaria the spleen is proportionately larger than the liver. In cases of amœbic abscess of the liver the spleen is not enlarged. If the spleen is enlarged in a case of hepatic abscess, this probably is embolic in origin. The fever in malaria is marked during the day, while in liver abscess the rise of temperature is seen in the evening. The blood picture in malaria is quite different from that of hepatic abscess, the former showing the plasmodium which is absent in the latter; in abscess there usually is a high leukocyte count with a corresponding increase of the polynuclear cells, which is not seen in malaria. The administration of quinine will generally clear up the diagnosis between malaria and liver abscess.

In cholelithiasis and cholecystitis and in chronic calculous obstruction of the common duct, the history will have great weight in making the diagnosis. In these conditions the symptoms point to conditions of the biliary tract rather than to the liver itself. The pyæmic abscess, complicating acute infection of the biliary tract, cannot always be recognized, although the increased area of tenderness over the hepatic region in liver abscess might cause one to infer that the liver had been infected.

Progonsis.—The prognosis in *traumatic abscess* will depend upon the character of the injuring force, the portion of the liver involved, and the virulence of the infecting micro-organism. The prognosis always is grave.

In *pyæmic abscess*, especially when multiple, the prognosis always is very grave and according to some writers, hopeless. Dieulafoy has stated that infection of the liver from acute appendicitis is always fatal. His statement has been challenged by Quénu and Mathieu who have analyzed fourteen cases which they claim are of this nature. The infectious focus was single, or there were only few foci, and operative measures cured twelve of the fourteen cases (page 166). When the foci are multiple the condition almost always results fatally. Personally, we have never seen recovery in a case of multiple abscess of the liver due to acute appendicitis.

In *amœbic abscess* death may occur in untreated cases from sepsis following perforation into the peritoneal cavity with peritonitis. Spontaneous cure may result if the rupture occurs into the pleural cavity or the lung, but very rarely if the rupture drains the abscess into the stomach or colon. Early operative interference always makes the prognosis more favorable. Coffin quotes Manson as having found a mortality of 57.7 per cent. in the Indian Army from 1891 to 1894. Of thirty-four cases analyzed by Coffin, sixteen died. Herrick lays great stress on the differential leukocyte count, in making the prognosis of these cases. He found that when the polynuclear count was below 80 per cent. the prognosis was good, the operative mortality being only 6.6 per cent. whereas in cases where the polynuclear cells were over 80 per cent. the operative mortality was 38 per cent.

Treatment.—The proper treatment of liver abscess is drainage. As pointed out by Herrick "the one vital necessity is that the abscess should be opened at its point of election, which would be the point where the abscess approaches nearest to the surface of the liver."

Two avenues of approach are open to the surgeon: one through the peritoneal cavity, and the other the transpleural route. The "combined operation," similar to that described in connection with injuries of the diaphragm (Vol. I, page 311) presents no advantages, and should not be employed. No attempt should be made to localize the abscess by means of the aspiring needle, as it is unreliable and fatalities have followed its use.[1] If the abscess cannot be clearly localized by the physical findings, an exploratory laparotomy should be performed. This will enable the surgeon to make a careful examination of the liver, and show him where the incision into the liver should be made. If accessible from within the abdominal cavity, the abscess may now be opened, after isolating the field of operation by gauze packs. In most cases, however, the approach by thoracotomy is to be preferred. If the needle is used for exploration it should be followed by immediate operation, before withdrawing it, whenever pus is found. However, we are firmly of the opinion that it is dangerous to explore before sufficient exposure has been obtained by a free incision. If the abscess cannot

[1] This practice was long in use as a therapeutic measure under the name of "hepatic phlebotomy" (Harley 1886).

be located readily after the abdomen has been opened, it may then be proper to resort to puncture of the liver with an exploring needle. Under such circumstances, as pointed out by Terrier and Auvray, this procedure can do no harm, and may do good, by relieving the hepatic congestion by means of what Harley called *hepatic phlebotomy*.

The transpleural route, employed in 1885 by Knowsley Thornton, gives the best access to the greatest area and should always be used unless the abscess presents anteriorly, or unless laparotomy is indicated for diagnostic purposes. (For description of operation see Chapter IX.) The transpleural route was employd by Israel, as early as 1879, in a case of hydatid cyst of the liver (page 197).

REFERENCES.

Bradshaw: Lancet, 1908, i, 146.
Coffin: Amer. Med., 1906, xi, 134.
Cyr: Traité des Maladies du Foie, Paris, 1887.
Dieulafoy: International Clinics, Phila., 1900, 10th Series, i, 144.
Douglas: Surg. Diseases of Abdomen, Phila., 1903, p. 288.
Ewald: quoted by Douglas, l. c.
Flexner: Amer. J. Med. Sc., 1897, cxiii, 553.
Harley: Brit. Med. J. 1886, ii, 899.
Herrick: Surg., Gynec. and Obstetr., 1910, xi, 472.
Jiminez: quoted by Heinemann: Virchow's Arch. f. Path. Anat., 1885, cii, 508. (Monograph dated 1858.)
Kehr: System of Practical Surgery, Ed. by v. Bergmann, Bull, et. al., Phila., 1904, iv, 639.
Kelly: Osler's Modern Medicine., Phila., 1908, v, 748.
Kieffer: Phila. Med. Jour., 1903, xi, 384.
Langenbuch: Chir. d. Leber u. Gallenblase (Deutsche Chir.), Stuttgart 1894, Bd. i, S. 269.
Loison: Bull. Soc. Chir., Paris, 1900, xxvi, 66.
Manson: Tropical Diseases, Lond. 1904, Rev. Ed. p. 437.
McDill: Journ. Amer. Med. Assoc., 1907, xlix, 491.
McWilliams: N. Y. Med. Jour., 1907, lxxxvi, 1054.
Melchior: Centr. f. d. Grenzgeb. d. Med. u. Chir., 1910, xiii, 161.
Munro: Annals of Surgery, 1905, ii, 692.
Musgrave and Clegg: Bull. Biol. Lab., Bureau of Science, Manila, 1904, xviii, 1.[1]
Quénu and Mathieu: Rev. de. Chir., 1911, xliv, 519.
Rhoads: Annals of Surg., 1904, i, 711.

[1] This work has not been accessible to us.

Rouis: Rech. sur. les suppur. endem. du Foie, Paris, 1860, p. 189.
Terrier and Auvray: Chir. du Foie et des Voies Bil., Paris, 1907, ii, 310.
Thornton: Brit. Med. J. 1886, ii, 903. (Transpleural operation for hepatic abscess.)
Waring: quoted by Langenbuch, loc. cit., S. 268.
Zancarol: Rev. de. Chir., 1893, xiii, 671.

Cirrhosis of the Liver.

Following the classification of Kelly, two main types of cirrhosis of the liver are recognized, *portal* and *biliary*. Kelly uses these terms, with subtypes, because in one the ætiological factor is "perhaps always transmitted by the portal circulation, the new-formed connective tissue is especially conspicuous in and about the portal spaces in the liver, and the obstructive symptoms are those of portal obstruction." The other is called biliary cirrhosis because the essential lesion is a radicular cholangeitis and the conspicuous clinical feature is jaundice, due to obstruction to the free flow of bile.

In *portal cirrhosis* the obtrusive symptoms above referred to are ascites, and gastrointestinal hemorrhages, the result of varices in the ultimate branches of the portal system of veins. It has been maintained by Hale White, Rolleston, and others, and Kelly concurs in this view, that patients with uncomplicated portal cirrhosis of the liver do not long survive the onset of ascites, rarely living long enough for more than one tapping to be necessary. These authorities claim that in patients who live and are tapped many times, either the diagnosis of portal cirrhosis is incorrect, or the condition is complicated by perihepatitis, chronic peritonitis, etc. It is only in this latter class of patients that the question of surgical treatment arises.

In cases of *biliary cirrhosis* there sometimes are accompanying lesions of the gall-bladder and bile-ducts, which may call for surgical treatment. A distinction is drawn by Kelly between true biliary cirrhosis so complicated, and cases of obstructive jaundice with chronic intrahepatic pericholangeitis, resulting in disseminated cirrhosis of the liver. A differential diagnosis is difficult except at the autopsy table or by prolonged study of specimens removed postmortem.

Surgical interference in cases of *portal cirrhosis* of the liver

has for its object the relief of ascites, or of recurring hemorrhages, and not the cure of the lesion in the liver. The operations advocated, therefore, must be considered as measures taken for the relief of symptoms and not as therapeutic measures inaugurated to cure the underlying disease. Little can be expected from operative treatment of cases of *obstructive jaundice* if this is postponed until hepatic cirrhosis has developed. *Surgical treatment of hepatic cirrhosis therefore is confined almost exclusively to relief of the ascites or hemorrhages which accompany the portal form of the disease.*

Ætiological Factors.—It usually has been taught, on the authority of Rolleston, that ascites in cases of cirrhosis of the liver is due to portal pressure and to toxæmia, the former causing conditions favorable to peritoneal effusion, and the latter interfering with the normal activity of the endothelial cells of the peritoneum. Portal obstruction alone is not the chief factor in the production of ascites, because the radicles of the portal vein lie nearer the mucous than the serous surfaces of the gastro-intestinal tract and dilatation of these radicles results in the production of varicosities; and these varicosities manifest their presence not by the occurrence of ascites, but by hemorrhages. Ascites is caused almost solely by changes in the endothelium composing the peritoneum; it is in the nature of a chronic serositis. In other words the toxæmia due to disordered hepatic function is a much more important cause of ascites than is the existence of portal obstruction. *But cases of portal cirrhosis sometimes are complicated by tuberculosis of the peritoneum, or by a chronic polyserositis associated with cardiac disease;* and in such cases it may not be the hepatic toxæmia but the complicating disease, which is responsible for the peritoneal effusion.

Under normal conditions the various anastomoses between the portal and systemic venous channels are able to care for the slight obstruction of the portal system. As stated by Deaver, "the radicles of the portal vein anastomose with the systemic veins in numerous places; among these anastomoses are the following: Radicles of the superior hemorrhoidal vein anastomose with branches of the middle and inferior hemorrhoidal veins, these last being tributaries of the internal iliac vein; the gastric tributaries of the portal vein anastomose with the lower œsopha-

geal veins, which empty into the azygos vein. In the suspensory ligament of the liver are veins which connect the portal system with the veins of the diaphragm; along the round ligament of the liver there are one or two veins which effect an anastomosis between the portal system and the veins of the abdominal wall."

When there is obstruction of the portal system, as is seen in cirrhosis of the liver,these anastomosing channels become enlarged to assist in establishing collateral circulation. The enlargement of the spleen, so frequently seen in connection with cirrhosis of the liver, is due to the damming back of the blood in the splenic vein as a result of the interference with the portal circulation. If the obstruction of the portal system cannot be cared for by the enlarged venous anastomoses, leakage occurs with repeated hemorrhages from the gastro-intestinal tract.

Most of the operative methods proposed for the treatment of ascites with cirrhosis of the liver seem to have been based on the theory that the ascites occurred as a direct transudate from the obstructed portal system. Many of the methods therefore are irrational, and if successful the happy issues have been due to factors not recognized at the time as essential. Vidal has well pointed out that efforts to relieve the ascites by attempts to establish a collateral circulation between the portal and systemic circulations are based on an erroneous idea of the pathogenesis of ascites. The quickest and surest way to establish such a collateral circulation is to make an anastomosis between the portal vein and the vena cava (Eck's fistula) thus draining the portal blood directly into the right heart. This delicate operation was successfully performed by Vidal (1903); he was forced to adopt it because he found there was no omentum available for epiplopexy, and as the patient was nearly exsanguinated from repeated hemorrhages, some method of relieving the portal congestion seemed imperative; but, though the hemorrhages were cured the Eck fistula did not prevent the recurrence of ascites, six weeks before death, which occurred four months after operation, and was due to an acute general infection evidently enterogenous. The liver, interposed as a filter to the hordes of microbes constantly absorbed by the portal blood stream, was side-tracked by the operation and these bacteria entered the general circulation with undiminished virulence; so that death from acute general infection must always

be anticipated under such circumstances. Another reason for condemning the operation, emphasized by Vidal, is that the withdrawal of the functions of the liver from the digestive and metabolic processes, necessitates an almost impossible restriction of diet.

The various methods of operative treatment proposed for cirrhosis of the liver have been summarized by Ricketts, as follows:

1. Incision through the abdominal wall, with temporary or permanent drainage of the ascitic fluid. A number of cases have been permanently benefitted by this procedure.

2. Paracentesis, or puncture through the abdominal wall, with temporary or permanent drainage. A number of recoveries have been reported, usually after a number of tappings. Lecreuz reported, in 1902, the history of a patient from whom he removed, during a period of five years, 1750 liters of fluid by sixty-five punctures; the patient remained well two years after the last tapping. The procedure, however, usually is useless except for temporary relief of the distress resulting from the pressure of the ascitic fluid.

3. Hepatotomy, or incising the liver to various depths after having first opened the abdomen, with temporary or permanent drainage.

4. Hepatotomy with a trocar, followed by temporary or permanent drainage. The results obtained are about the same as with hepatotomy with the knife.

5. Cholangeiostomy. Thornton in 1887 succeeded in draining the biliary tract by penetrating the right lobe of the liver to one of the larger branches of the hepatic duct, in which calculi were lodged. Ricketts suggests a modified cholangeiostomy performed by cutting through the lobe of the liver and pulling the gall-bladder through the lobe as far as possible. The gall-bladder is then anchored so that it will become united to the liver substance. Subsequently the viscus is to be opened and drained externally. It is not clear just what this operation is intended to accomplish.

6. Cholecystenterostomy has been performed by Combenale and Dubar for the purpose of effecting better drainage.

7. Cholecystostomy was performed by Delagenière in 1901 for cirrhosis and this method of treatment was fully discussed by his pupil Bernard.

8. Injection of caustics into the peritoneal cavity has been

resorted to on the theory that the irritation produced will cause increase in the activity of the peritoneum.

9. Ligation of the portal vein has been performed for the purpose of lessening the congestion of the liver and encouraging the establishment of a collateral circulation. The procedure was described by Pascale in 1901.

10. Hepatopexy (page 163) was performed by Delagenière in 1897. Ricketts reports two of his own cases of cirrhosis in which the same operation was performed.

11. Epiplopexy or omentopexy, variously described as the Talma, the Talma-Drummond, or the Morison operation, has been employed oftener than any other procedure devised (see below).

12. Eck's fistula, which is made by establishing a communication between the portal vein and the vena cava. Vidal's (1903) operation of this nature has already been discussed.

13. Splenopexy, or anchoring the spleen to the anterior peritoneal wall, with or without omentopexy, has given some very favorable results.

14. Multiple visceropexy is suggested by Ricketts as a procedure that might accomplish much good.

We believe that in seeking operative relief for the symptoms of hepatic cirrhosis, the surgeon always should bear in mind what the two chief symptoms are—ascites and hemorrhages; and that remembering the probable pathogenesis of each of these symptoms he should adopt his plan of operation accordingly. If the hemorrhages are the predominant feature, measures for establishing a collateral circulation are indicated; if there are no hemorrhages, but ascites is annoying, it is probable that complete evacuation of the fluid by laparotomy, with such alteration in the nutrition of the serous surfaces of the peritoneum as accompanies this simple operation, will be as effective in relieving the ascites as will any more complicated procedure. We quite agree with Bogojawlensky who claims that the benefit of the operation of omentopexy is due to the laparotomy and consequent hyperæmia rather than to the fixation of the omentum, although the latter may help. He induces as much hyperæmia as possible during the operation. Bogojawlensky also claims that it is essential that all ascitic fluid be removed from the peritoneal cavity on account of

the danger of too rapid absorption if the kidneys are not functionating properly. Dock also is of the opinion that the relief of the ascites may be due more to the operation on the serous membrane than to the opening of collateral circulation.

The most efficient method for the **establishment of a collateral circulation** is **epiplopexy** (omentopexy). Talma began his studies on this subject in 1889; but the first case in which the operation was successful was published by Drummond and Morison in 1896. We do not believe that this operation is indicated in cases of cirrhosis unaccompained by gastro-intestinal hemorrhages; and when its performance has been followed by the disappearance of an uncomplicated ascites, it is highly probable, as noted above that the success was attributable to other factors in the operation and not to the epiplopexy itself.

Complication of cirrhosis with nephritis does not necessarily contraindicate Talma's operation; but any operation is contraindicated when the patient is markedly weakened by disease of the heart and kidneys; and is absolutely contraindicated when the functional activity of the liver cells has been abolished, as shown by the presence of urobilinuria and acholia. Long-continued jaundice acts as a contraindication on account of the predisposition in these cases to postoperative hemorrhage.

The results of the operation of epiplopexy, undertaken for the relief of ascites in cirrhosis of the liver may be seen from the figures collected by Ricketts: he notes 1565 cases in which the operation of epiplopexy was performed, the results being as follows:

Patients cured	30.4 per cent.
Patients relieved	19.8 per cent.
Patients unrelieved	39.2 per cent.
Patients died	10.6 per cent.

McWilliams quotes the following statistics:

Author.	Cases.	Symptomatic Relief.
Koslowsky	168	46 per cent.
Greenough	105	42 per cent.
Monprofit	224	35 per cent.

Bunge reports 33 per cent. of permanent cures and 33 per cent. improved.

The report of Dock illustrates the usual course of those cases which are ultimately cured of the ascites. In this case tapping was resorted to fourteen times and was followed by omentopexy, performed in 1902 by Edward Hamilton of Houston, Texas; during the following seven months paracentesis was performed ten times for the relief of the ascites. These tappings were followed by permanent relief, internal medication fully controlling any slight subsequent recurrence of the ascitic fluid during the seven years which had elapsed since operation up to the time of Dock's report in 1909.

The *technique of the operation* is discussed in Chapter IX.

For the **relief of the ascites,** as already pointed out, it is more rational to resort to laparotomy, with sponging of the parietal peritoneum and of that covering the liver; or even a resort to hepatopexy as practised by Delagenière. The performance of epilopexy at the same time is not to be condemned, since it may be of benefit in relieving unrecognized gastro-intestinal varices.

REFERENCES.

Bernard, E.: Thèse de Paris, 1901.
Bogojawlensky: Zentralbl. f. Chir., 1909, xxxvi, 292.
Deaver: Surgical Anatomy, Phila., 1903, vol. iii, p. 189.
Delagenière: Arch. Prov. de Chir., 1904, xiii, 646.
Drummond and Morison: Brit. Med. Jour., 1896, ii, 728.
Dock: Jour. Amer. Med. Assoc., 1910, i, 275.
Kelly: Osler's Modern Medicine, Phila. 1908, v, 758, 777.
Koch: Deuxième Congres de la Soc. Internat. de Chir., Bruxelles, 1908; in Journal de Chir., 1908, i, Suppl., p. 62.
Lecreuz: Rev. Gén. de. Clin. et de Thérap., 1903, xvii, 215.
McWilliams: N. Y. Med. Jour., 1907, lxxxvi, 1053.
Monprofit: Assoc. Française de Chir., Congrès xvii, 1904, p. 24.
Morison: see Drummond and Morison.
Pascale: Riforma Medica, 1901, xvii, Part 3, 530, 543, 554.
Ricketts: N. Y. Med. Jour., 1909, xc, 533.
Rolleston: Osler's Modern Med., Phila., 1908, v, 500.
Talma: Report by Lens: Nederl. Tijdschr. v. Geneesk., 1892, 2 R., xxviii, i, 645.
Thornton: Brit. Med. J. 1887, ii, 1148.
Vidal: Assoc. Française de Chir., 1904, xvii, 183.

Cysts of the Liver.

The most common cyst of the liver is the **hydatid** or **echinococcus.** Though knowledge of hepatic cysts dates back to the times of Hippocrates and Galen, and though the anatomists of the sixteenth and seventeenth centuries accurately described such cysts, in those times the cysts were supposed to be enlargements of the lymphatics. Pallas (1760) proved that the cysts were independent parasites, and also showed the close relation they held to the tape-worm. Bremser, in 1819, published the first accurate account of the echinococcus occurring in the human liver. The subject was thoroughly discussed by Davaine in his treatise on intestinal parasites, first published in 1860.

The exciting cause of the echinococcus cyst is the *Tænia echinococcus* (*Echinococcus granulosus*), a parasite found in the upper intestinal tract of several animals, such as the dog, the wolf, and the jackal. Richardson, in 1867, and later Madelung (1885) have shown that the echinococcus is also found in sheep. Kehr states that the domestic cat and rabbit may also be the source of infection.

The ova enter the gastro-intestinal tract of man with food or drink, or possibly as the result of handling or being licked by an animal infested by the parasite. The capsule is digested in the intestinal tract, and the embryo is liberated. In the larval state the echinococcus is globular in form. It possesses six hooklets and four suction discs which aid it in boring into the tissues. It finds permanent lodgment in various portions of the body; in the liver, spleen, kidney, lungs, etc., the liver being the most frequent site of its lodgment and development. It is probable that the larvæ enter the radicles of the portal vein and are then carried directly to the liver. Some may also reach the liver through the bile-ducts. Douglas quotes Davaine as having found the echinococcus in the liver in 166 of 376 cases; Böcker, in twenty-seven of forty cases; and Weisser in 451 of 900 cases. The ova as a rule do not escape again from the human body, though they are discharged constantly from the bodies of sheep in the slaughter house, to be devoured by dogs and again developed into tape-worm (Morris). The proper means of *prophylaxis* of

hydatid disease are thus indicated, in guarding the food supply of dogs, and in proper attention to their dejecta.

Age and Sex.—Echinococcus disease is seen most frequently in those between the ages of twenty and thirty years. It may be found at any age; even in the fœtus the cyst has been found of such size as to prevent delivery. It has no predilection for either sex.

Distribution.—Echinococcus disease is found most frequently in Iceland. In that country the close association with dogs is marked, and this fact is held accountable for its frequency. According to Morris one-seventh of the human mortality in Iceland is due to hydatids. The multi-locular cyst rarely is seen in Iceland. In Australia and Italy the disease is not uncommon. France and Germany are rather free from it, while in England and the United States it is comparatively rare.

Pathology.—After the parasite (in larval state) reaches the liver it loses its hooklets and enters the immature or cysticercus stage. Inflammatory changes cause a protective connective-tissue encapsulation. The cyst-wall consists of *two layers*, an outer laminated membrane or capsule; and an inner vascular layer variously designated as the parenchymatous, granular, or germinal layer. The contents consist of a clear, colorless, transparent fluid, non-albuminous, of a specific gravity of from 1000 to 1015. It contains sodium chloride and traces of succinic acid and of sugar. When the capsule is broken down either by erosion or suppuration, or when the cyst becomes infected with pyogenic bacteria, the fluid becomes turbid; sometimes it is bloody or bile-stained. Boinet in studying hydatid fluid, extracted a ptomaine from it in the form of a prismatic crystal, fern-leaf in shape. The death of a mouse occurred five minutes after the administration of three-sixty-fourths of a grain under the skin. A larger dose given to a rabbit caused convulsions, alteration in the rhythm and rapidity of the respirations, tachycardia, dilated pupils and collapse, the symptoms usually assigned to hydatid intoxication. Fowler states that this toxin is much more abundant in cases in which puncture and electrolysis have transformed the clear fluid into a turbid syrupy fluid which is rich in albuminoid matter.

When the cyst is fertile, daughter cysts develop within the

original, or parent, cyst; other, or granddaughter cysts, at times develop within the daughter cysts. The heads or scolices of the parasites are found on the inner surface of the germinal layer, in pedunculated vesicles called by Prudden "Brood-capsules." The walls of these vesicles are similar to those of the primary cyst. A single scolex or several scolices may be found in each of the brood-capsules. They are like the parent parasite, having the same number of hooklets and suction discs. The scolex may be free in the capsule; if the capsule ruptures, the scolex will then be found free in the cyst. In some instances deposits of lime salts will be found in the scolex.

After the cysts have undergone degenerative changes, the hooklets and portions of the cyst membrane may be found in the resulting detritus. Where calcification has taken place, it often will be difficult to recognize or determine the causes or origin of the cyst.

Prudden cites two rare forms of echinococcus cysts, in one of which, *echinococcus scoleciparíens*, the secondary vesicles are formed on the outside of the primary cyst-wall. The second variety, *echinococcus multilocularis*, which is more common in man than the former, results from disturbances in the development of the cysts. In an encapsulated mass, series of irregular cysts will be surrounded by bands of connective tissue of varying widths. This has been called by Vierordt "echinococcus alveolaris." A cross-section of the tumor gives an appearance which accounts for the term "alveolar colloid" which was formerly applied to it.

Thompson exhibited an interesting specimen of multiple hydatids. "The liver was the seat of extensive growths, there being five distinct tumors in various stages of activity. Two had suppurated, two had died and had been converted into inert masses, while the fifth was in the active growing stage." Multiple cysts occur in about 12.5 per cent. of cases. Their existence may be suspected at operation if evacuation of the first cyst does not cause a sufficient diminution in the size of the liver.

Echinococcus cysts of the liver generally form in the right lobe, and in about 90 per cent. of the cases the cyst is solitary. Any part of the liver may be involved. The size and shape of the organ vary with the position of the cyst. If it is in the centre of

the liver there generally is a more or less uniform enlargement; if near the border or on the lower surface of the liver, or if there are multiple cysts, the shape of the liver is greatly altered. Cysts on the lower surface generally grow downward and ultimately may fill the greater part of the abdomen, even reaching the pelvis, as in a case reported by R. S. Fowler. When growing on the upper surface of the liver, the pressure against the diaphragm may be great enough to compress the overlying lung. Usually a tumor is noticed in the right hypochondrium or in the epigastrium.

Echinococcus disease may last for years. The growth of the cyst is slow, the course of the disease extending over a period of from two to thirty years. It may exist for years without giving rise to symptoms and may be discovered or suspected only after the abdomen has been opened at autopsy. In an analysis of twenty cases, Barrier found that in three the disease had lasted two years; in eight, from two to four years; in four, from four to six years; and in the remaining fifteen cases the duration was fifteen, eighteen, twenty and even thirty years. In fourteen cases studied by Henry Morris, the average duration of symptoms was about seven years. In the series of cases studied by Cauchoix, the probable duration of the disease before treatment was sought is indicated in fifteen instances: this period varied from a few days to thirty-six years (Obs. xxxvi), the average duration of symptoms being more than five years.

The *untreated hydatid* may progress indefinitely, death from another cause intervening. Death may result at any time from rupture as a result of trauma, or from infection with suppuration; or spontaneous rupture without infection may occur. In cases where the parasite dies, or ceases to grow, the cyst usually atrophies, and the contents of the sac resemble sebaceous matter, or are greatly altered by calcareous changes. A *spontaneous cure*, of course, results. If the cyst opens into the *biliary apparatus*, sterile bile as a rule will kill the parasite and effect a cure. If the bile is infected, however, suppuration of the cyst will ensue. *The cyst may rupture spontaneously* into one of the hollow viscera, such as the stomach or intestine, into the lung or pleural cavity, or into the free peritoneal cavity. When the stomach is invaded the contents of the cyst will be vomited; if the lung is the organ involved, violent coughing will cause the expulsion of the fluid, and some-

times of unbroken daughter-cysts; if rupture has taken place into the intestine, the contents of the cyst may be recovered in the stools. When rupture takes place into the general peritoneal cavity, the result will depend upon the condition of the cyst contents: if there is infection present, a suppurative peritonitis will follow with a mortality of about 90 per cent.; if the contents are sterile, multiple cysts may form in the peritoneum, unless the patient succumbs to the septic condition caused by the absorption of the ptomaines and toxines contained in the fluid. A diagnostic point of some value in these cases is the urticarial rash which develops rather rapidly. Rupture of the cyst may be external in rare cases; occasionally it ruptures both internally and externally. Dévé describes **hydatid gaseous cysts** (*Pneumokystes hydatiques*) of the liver, a condition first mentioned by Lænnec; the condition is attributed to rupture of the echinococcus cyst into the stomach, intestine or lung; rarely it is caused by putrefactive changes within an unruptured cyst. The term is also applied to an hydatid cyst to which air has been admitted as the result of operative procedures, thus being analogus to the term "open pneumothorax." Dévé collected fifty-one cases of hydatid pneumocysts; operation was done in fourteen cases with eight deaths resulting.

Symptoms.—Echinococcus disease may extend over years without giving rise to symptoms. Unless there is infection of the cyst, the symptoms, as a rule, will be limited to the effects of the pressure exerted by the tumor. At times the patient may complain of a dull, dragging feeling, with some discomfort and slight pain in the epigastrium, loin, or back. When the cyst is large and situated on the upper surface of the liver, pressure on the diaphragm may cause interference with respiration. In such cases the liver will be pushed downward. When the cyst grows large enough to interfere with the normal functions of other organs, pressure symptoms referable to the involved organs will be noted. Pressure on the biliary ducts may cause jaundice; pressure on the inferior vena cava will cause ascites or œdema of the lower extremities; pressure on the portal vein will cause gastro-intestinal varices or ascites; pressure on the gastro-intestinal tract will cause functional disturbances. Attacks of urticaria are not uncommon.

When large enough to be palpable, the connection of the tumor with the liver is evident; its surface is smooth and rounded, im-

parting to the examining fingers the sensation of a dense fluctuating mass. When the tension within the cyst is high, fluctuation is not present and the tumor seems a solid mass. A jelly-like feel is absent in the great majority of cases; Finsen was unable to detect it in a series of 235 cases. If adhesions are present, the tumor is more or less fixed; if absent, the tumor moves with respiration. There is no tenderness on pressure, as a rule, unless the cyst is infected.

Hydatid fremitus, a tremulous impulse felt in palpation over a hydatid cyst, may or may not be elicited. It is supposedly caused by the impact of the daughter-cysts against one another. If, however, the parent sac contains fluid the daughter-cysts will be suspended and will not give this sign.

A sign that is considered pathognomonic of hydatid disease by Santoni was described by him in 1894, when he found that ausculatory percussion revealed a peculiar sound, or booming, of low tone, lasting but a short time and ceasing abruptly. The mass is dull on ordinary percussion.

After *infection of an echinococcus cyst* has taken place, the symptoms presented are much more intense and generally alarming. The patient becomes septic, with irregular temperature, abdominal pain and marked tenderness over the region of the tumor. Symptoms of peritoneal irritation usually are marked.

The symptoms presented in echinococcus disease are often very misleading, as may be seen from the following case history:

L. E., female, aged twenty-two. Born in Russia; hat-trimmer. Admitted to the German Hospital, December 14, 1909. Family history negative. No tuberculosis. No carcinoma. Has been in this country five years. Well until present illness. No acute diseases, no operations. Habitual constipation. Menstrual history surgically negative.

Present Illness.—Began to "feel badly" two days before admission, complaining of indigestion which she had had at irregular intervals during past three months. Her attacks of indigestion began about two hours after meals, and were characterized by a sensation of discomfort and bloating in abdomen. No nausea or vomiting. Present attack began with pain in epigastrium, a little to right of median line, midway between costal margin and umbilicus. This has remained the most acutely painful point, but pressure over any portion of the abdomen caused pain. Patient had never been jaundiced.

Physical Examination.—Well-developed and well-nourished girl, expression worried. Face flushed, cheeks hectic. Knees kept flexed. Respiration

costal. Head negative. Tongue slightly coated. Chest and heart negative. Abdomen full but not distended. Abdomen generally rigid and in upper portion hard. Tender everywhere. Liver dullness completely obliterated. No mass palpable. No marked dullness in flanks. Peristalsis present. Pelvis normal with exception of tenderness on pressure throughout vaginal vault. Pulse rapid but regular, volume small, tension high.

Hb. 74 per cent.; leukocytes, 24,550; polynuclears, 88.5 per cent.

Provisional diagnosis of duodenal ulcer with perforation was made, and operation advised.

Operation on day of admission. Ether anæsthesia. Upper right rectus incision. Intestines distended. Stomach, gall-bladder, duodenum and pancreas apparently normal to palpation and inspection. Palpation of liver revealed a nodule situated in the left lobe on upper surface, anteriorly. Area of yellow-white tissue protruded from surface, about the size of top of wine-glass. Incision made into tumor, through very tough wall. Cavity about size of lemon revealed. Cavity filled with echinococcus daughter-cysts, not infected. Cavity thoroughly curetted and packed with gauze. Wound closed around drainage.

December 27, 1909: Course of convalescence normal. All gauze removed to-day. Cavity in liver granulating.

December 28, 1909: Patient up and around ward, talking with other patients. Complained of sudden sharp pain over heart, with sudden cessation of heart action and of respiration. Was dead within four minutes.

Autopsy revealed a second cyst about the size of a lemon situated in the right lobe of the liver. No evidence of peritonitis. No demonstrable cause of death.

Diagnosis.—It is practically impossible to diagnose hydatid cysts of the liver, when the tumor is small. When the cyst is palpable, its evident connection with the liver, and its generally rounded, smooth surface are suggestive features. Unless fastened in place by adhesions, it moves with respiration. A clear history of the course of the disease from the first symptom presented usually helps to clear up the differential diagnosis. When the cyst has become infected the diagnosis is comparatively easy, especially in those cases where echinococcus disease is known to exist. There will be marked pain, an irregular temperature, and symptoms of peritoneal irritation in addition to those formerly presented by the disease. These symptoms may be less marked, as pointed out by Kehr, when the tumor becomes adherent to the abdominal wall and threatens to break down the skin. The diagnosis must then be made from the redness and the peculiar condition of the skin and subcutaneous tissues, together with the general symptoms presented.

In making a correct diagnosis of hydatid cyst, the usual causes of tumors of the liver must be considered and excluded. Carcinoma, abscess, syphilis and tuberculosis are the underlying causes of most liver tumors. Sarcoma, lymphadenoma, angeioma, myxoma, fibroma, and atheroma are also observed, but not so frequently. Tumors of the adjacent organs must also be considered. Those most likely to throw doubt on the diagnosis are tumors of the gall-bladder, of either kidney, or of the spleen. Empyema and subphrenic abscess may also be confounded with hydatid cyst.

Primary carcinoma (page 211) of the liver is rare. Usually carcinoma is secondary to a similar growth in some other organ; the presence of carcinoma in a locality other than the liver makes the diagnosis of carcinoma of the liver, when a tumor of that organ is discovered, most probable. The carcinomatous tumor is more irregular than the cyst, presents a roughened surface, and feels harder than the cyst. There is increasing pain in the region of the liver, with decrease in weight, loss of appetite and of strength. Emaciation soon becomes marked, while the liver shows increase in size, and becomes tender to the touch. Jaundice may be early or late in the disease and always increases in intensity.

In *abscess of the liver* (page 164) a palpable tumor may develop either in the epigastrium or beneath the right costal margin. It is tender on palpation, usually increases rapidly in size, and gives marked constitutional symptoms, such as chills, sweating, irregular temperature, and a peculiar sallowness of the face. The previous history usually shows an antecedent focus of pus in some other region of the body, especially in the appendix, or the occurrence of dysentery. The stools should be carefully examined for amœbæ; a blood examination usually shows a marked increase in the white cells. This is not the case in an uninfected hydatid cyst. The differential diagnosis between an infected suppurating hydatid cyst, and an abscess of the liver often is extremely difficult, and depends more upon the clinical history than upon the physical examination.

Syphilitic tumors (page 208) of the liver are much more common in this country than hydatid cysts. In the cases of recent gummata, there usually is a distinct tumor, either in the form of a hard nodule or a large flat mass. Usually a clear history of spe-

cific infection can be obtained. In every instance where syphilis of the liver is suspected, potassium iodide should be administered. This will cause some diminution in the size of a syphilitic tumor, although it may not entirely disappear. The Wassermann test also should be made.

Tuberculosis of the liver at times may cause a distinct tumor, as shown in the case reported by MacKenzie (Douglas). In this case there were multiple abscesses of the liver, with a globular swelling about the size of an orange in the right lobe. In such instances, the symptoms presented are similar to those of chronic abscess of the liver, with acute exacerbations.

The other tumors of the liver seldom are seen and do not present symptoms which would allow a differentiation from the non-infected small hydatid.

In *empyema* and *subphrenic abscess*, there usually is a history of pneumonia, pleurisy, cholelithiasis, cholecystitis, appendicitis, or some other lesion which leads to a correct differentiation between the condition present and an echinococcus cyst.

Ghedini claims that the presence of an echinococcus cyst in the body may be revealed by the *hæmolytic blood test.*

Treatment.—Other things being equal surgical treatment is indicated in every case of hydatid cyst of the liver as soon as the diagnosis is made. There is nothing to be gained by waiting, and medicaments are totally powerless to destroy the worm. It will be convenient to study first the treatment of the simple, non-infected, hydatid cyst of the liver; and then to discuss the proper treatment of the various complications that may arise (suppuration, rupture, etc.). The methods for prophylaxis have already been referred to (page 189).

Method of access for evacuation of an echinococcus cyst deserves a few words. Most cysts develop downward and are best exposed by laparotomy; resection of the costal border or division of the suspensory ligament may be necessary for better exposure. For those growing upward beneath the diaphragm the operation is similar to that for the drainage of an abscess of the liver by the transpleural route (Israel). Roser claims that he proposed this plan as early as 1864.

Treatment of Single Uncomplicated Cysts.—*Puncture or aspiration* of the contents of the cyst was the earliest form of

surgical treatment adopted. The fact that cures were reported from these simple methods merely shows that the patients were not kept under observation long enough after operation to exclude the possibility of recurrence. This may not take place for many months, or even years. If infection occurred, either before or after aspiration, it was recommended to open the cyst widely and drain it; under such circumstances it usually was adherent to the abdominal wall. The method of opening and drainage appears first to have been erected into a principle, applicable to all echinococcus cysts of the liver, by Landau in 1880; the procedure is termed marsupialization of the cyst, that is, converting it into a pouch.

Marsupialization.—The cyst is exposed by laparotomy, its walls are sutured to the edges of the abdominal incision, and it is opened then or at a subsequent operation. Its contents are evacuated, including detached daughter-cysts, and such scolices as have escaped from their brood-capsules; the inner capsule of the cyst (germinal layer) is also removed. The outer capsule should not be disturbed; it is closely surrounded in most cases by dilated biliary channels (perhaps containing infected bile) and by blood-vessels (especially portal or suprahepatic veins) of unknown size which may give rise to troublesome or even fatal hemorrhage. The cyst having been thus emptied, its cavity is stuffed with gauze, and allowed to heal by granulation. Though the immediate mortality of this operation is low (Cauchoix found ten deaths among 185 operations recorded by Végas and Cranwell), it has manifest disadvantages in the form of postoperative complications, which no longer commend it to surgeons. Chief of these objections is the long period of convalescence: Cauchoix found that though the great majority of cysts were completely closed in from one to four months, yet that in many patients the fistula continued to discharge for six months or a year. Moreover, it is very difficult to prevent secondary infection when marsupialization is employed, the case being analogous to that of a cold abscess opened and drained; and suppuration in an opened hydatid cyst may become a very serious matter. The secondary discharge of bile which often occurs through the fistulous tract may seriously impair the patient's health. These biliary discharges as well as spontaneous hemorrhages into the opened

cyst, are attributable to the negative pressure created in the cyst by opening and drainage.

Suture of the incision in the cyst, after the evacuation, and closure of the abdominal wound, was a method introduced in 1883 by Knowsley Thornton and popularized in 1891 by Bond. The sutured incision in the cyst-wall was fixed to the abdominal wound. In this way it was hoped to avoid the disadvantages attendant upon prolonged drainage, at the same time permitting secondary opening and drainage of the cyst should occasion demand.

Reduction of the evacuated cyst, without suture of the incision in its walls, and closure of the abdominal wound without drainage, was employed by Ryan and by Hamilton Russell in 1894 and adopted by a few other surgeons; but the dangers of secondary infection of the peritoneum through bile or blood effused into the cavity of the unsutured cyst soon caused this method to be abandoned.

Endocystorrhaphy (*Capitonnage*) was adopted in 1896 by Delbet and by Posadas. They diminished but did not entirely obliterate the cavity of the cyst, after its evacuation, by interrupted sutures, cautiously passed through its walls. The cyst was completely closed, and was not fixed to the abdominal wound, which was sutured without drainage.

Enucleation of the cyst from the surrounding hepatic tissue has been employed in a few cases; but the danger of opening into blood or bile-channels renders it a most unsuitable method. Cauchoix refers to fourteen cases, in four of which the operation could not be completed.

Extirpation of the cyst may be done when it is pedunculated; or a small portion situated within the liver may be removed by partial hepatectomy. Fowler prefers this to any other method, whenever practicable. Its advantages are a shortened convalescence, freedom from a biliary fistula, and assurance that the entire disease has been removed.

Prophylactic Treatment (*Formolization*).—Under this title, Cauchoix describes a method of sterilization of the cyst contents by injection of a 1 per cent. formalin solution with the view of preventing recurrence of the disease. It was demonstrated experimentally by Dévé (Thèse de Paris, 1901) that each of the

parasitic elements contained in echinococcus cysts is capable of reproducing the primary lesion; and Cauchoix showed that such recurrence occurred clinically, though often not for years after the primary operation. After various other antiseptics had been tried with no very marked success, Quénu (1902) adopted formalin solution (1 per cent.) as the sterilizing fluid, and employed the following technique in a number of cases with marked success: The cyst was exposed by laparotomy, and thoroughly isolated by gauze packing. The fluid contents of the cyst were then withdrawn through a trochar and canula by syphonage into a funnel. To avoid possibility of soiling the surrounding tissues, a very fine canula was employed and the trochar was passed through the wall of the rubber tubing attached to the end of the canula, so that when it was withdrawn no leakage occurred in its tract. When the cyst has been thus evacuated, the funnel is emptied, and is then filled with the formalin solution which is allowed to enter and distend the cyst, by the force of gravity. This solution is left in the cyst for five minutes, and is then withdrawn. The cyst, thus sterilized, is incised freely, and the germinal membrane is removed. Repeated laboratory examinations of this membrane showed that all parasitic elements had been killed by the formalin solution. The cyst therefore may be closed with impunity, restored to the abdomen and the external wound closed without drainage. It always is well, however, to fix the cyst-wall to the abdominal wound so that should an intracystic effusion of bile or blood demand evacuation this can be accomplished without wide opening of the peritoneal cavity.

This *prophylactic treatment of Quénu* undoubtedly is the best method of operation in all cases to which it is applicable; but there are a few to which it is not suited: cases, for example, when the hydatid cyst is full of daughter-cysts and contains no fluid, so that it cannot be evacuated by puncture. In such circumstances Cauchoix recommends that the surrounding tissues be protected by gauze soaked in formalin, so that the parasitic elements unavoidably discharged over the field of operation may be promptly killed.

Treatment of Complicated Cysts.—This subject has been well studied by Cauchoix and we have made free use of his valuable memoir in what follows.

Suppurating hydatid cysts require the same treatment as abscesses of the liver; but when the infection is of very low grade, as manifested by the clinical history, it may be possible to close the cyst completely after the use of a formalin injection, or at least to suture the incision tightly around a drainage tube which may be removd in about four days. According to Végas and Cranwell the *mortality* in cases of suppurating cysts treated by marsupialization is from 20 to 30 per cent.

Rupture of a cyst into the peritoneum requires immediate laparotomy and drainage, both of the cyst and of the pelvis. This is true not only in the case of rupture of a suppurating cyst, but also when an aseptic cyst ruptures, because in the latter case the secondary development of peritonitis from effusion of bile (choleperitonitis) is much to be feared.

Rupture of a cyst into the intestine is dangerous because of the secondary infection of the cyst which is nearly sure to occur. The proper treatment is laparotomy so soon as the immediate shock of the accident subsides, with repair of the intestinal defect and drainage of the cyst. Rupture occurs oftenest into the duodenum, transverse colon or stomach.

Compression of the biliary ducts by an echinococcus cyst leads soon to a condition of angeiocholitis, which demands choledochotomy and hepaticus drainage in addition to the treatment appropriate to the cyst itself.

Rupture of a cyst into the biliary passages is by no means rare. It was carefully studied by Quénu and Duval in 1906. In most cases this occurrence is attended by severe pain, and the appearance of septic symptoms, chills and fever. Unless the existence of a hydatid cyst is already known, the recognition of such a complication would be very difficult. The common duct may be obstructed by the impaction of some of the cyst contents, or merely by inflammatory swelling. Usually choledochotomy and hepaticus drainage will be required, in addition to proper treatment of the cyst. In six operations in which cholecystostomy was done, with drainage of the cyst, there were two deaths. In eight operations on the common duct there were three deaths (Quénu).

Rupture of a cyst into the thoracic cavity is another very fatal complication. Even if it ruptures only into the *pleura*, symptoms of pulmonary distress are usual; the pleural effusion is bile stained

(cholethorax), owing to leakage of bile into the cyst cavity. Cauchoix collected five cases, with three deaths. The proper treatment is pleurotomy, with free drainage, and marsupialization of the cyst. If rupture into the *lung* or *bronchial tubes* occurs, death is almost inevitable. Treatment is the same as for pulmonary abscess; the hepatic cyst should be drained also, preferably through the thoracic wound.

The mildly infectious nature of some suppurating hydatid cysts is exemplified in the following case, in which the cyst was opened and drained without infecting the general peritoneal cavity.

M. B., male, aged twenty-two, born in Austria-Hungary, admitted to the German Hospital (Medical Department), October 15, 1909. Came to the United States three years ago, since which time he has been employed as a butcher. Denies venereal infection. Family history negative. Had attacks similar to present, eight years ago. Has had no other illness of any kind.

Present illness began two weeks before admission. Had sudden attack of severe abdominal pain, greater in epigastrium and radiating across upper abdomen and into back. Pain severe and lasted several hours. There was some epigastric swelling. Vomited after eating. Took castor oil and felt better. Had second attack five days before admission, with vomiting. Third attack began day before admission, and lasted two hours. Vomited after drinking glass of milk. During attack of pain felt hot and cold alternately and had a slight chill. Appetite remained good. Bowels fairly regular until lately.

Physical Examination.—Fairly well-nourished man, face covered with acne. Tongue heavily coated in middle and back. Teeth in fair condition. Head, lungs and heart negative. Abdomen flat. Liver extends from fifth interspace to 6 cm. below costal margin. Edge rounded. In epigastrium and slightly to left is a mass with rounded edges, firm in consistence, tender on pressure. Moves with respiration. Spleen not palpable. No rose spots. Extremities negative.

October 16, 1909: Yellowish-brown, liquid stool. Occult blood absent. Trace of bile. Alkaline reaction. Soaps, neutral fats and fatty acid crystals. Many yeast cells. Few epithelial cells.

Stomach test-meal: Semi-liquid stomach contents, not completely digested. Acid in reaction. Free hydrochloric acid, sixty-four. Total acidity, ninety. Occult blood absent. Bile absent. Starch, neutral fats, epithelium, yeast cells present.

X-ray examination showed shadow to right of median line extending from right costal margin half way to umbilicus, affected slightly by deep inspiration. Continuous with liver shadow.

October 20, 1909: Hæmoglobin, 95 per cent.; leukocytes, 11,200.

October 26, 1909: Temperature, 99°; respiration, 24; pulse, 84.

October 27, 1909: Temperature, 103°; respirations, 26; pulse, 104. Patient seized with attack similar to those described. Severe pain in upper abdomen radiating to back followed by rise of temperature and pulse rate. Swelling in epigastrium very tender. Has persisted and extended more to right. Leukocytes, 16,500. Polynuclears, 84 per cent.; lymphocytes, 8.5 per cent.; mononuclears, 1.5 per cent.; eosinophiles, 2 per cent.; basophiles, 0 per cent.; transitional, 3.5 per cent.; unidentified, 0.5 per cent.

Transferred to surgical ward. Patient lies on side with head down and knees drawn up. Swelling in epigastrium visible, about size of an orange, exceedingly tender, dull on percussion, evidently continuous with liver. Moves very slightly with respiration.

Operation by Dr. Deaver, October 30, 1909: Ether anæsthesia. Upper right rectus incision. Left lobe of liver found lower than usual, the convexity being boggy. Few adhesions between left lobe and surrounding structures. In liberating the adhesions, wall of a purulent collection in the left lobe was ruptured and thin stream of pus was liberated. Attempts at aspiration were futile. Surrounding region well protected by means of gauze pads, mass incised and large quantity of pinkish-yellow faintly odorous fluid liberated. The fluid contained many white cyst-like bodies in a state of collapse, resembling white grape skins, varying in size from a small pea to a bird's egg. About a pint of material removed. A cavity about the size of a large fist remained. This was curetted carefully, and then drained with large rubber tube and gauze packing. Wound closed to edge of drainage.

November 12, 1909: All gauze removed under primary chloroform anæsthesia. Gauze packing introduced.

November 22, 1909: Gauze removed, followed by cyst-wall.

November 26, 1909: Cavity granulating. Wound discharging considerable bile.

Discharged from hospital December 24, 1909, with small sinus. No discharge of bile.

Temperature ran an irregular course, until the nineteenth day after operation, when it reached normal and remained so.

Non-parasitic cysts of the liver may be divided into two classes, the *congenital* and the *acquired*. Among the former are dermoid cysts and multiple epithelial cysts. The former are operable. The latter are of interest mainly from a diagnostic point of view, as they are inoperable and a fatal result usually occurs from the "cystic degeneration" of the liver, which is frequently found associated with cystic degeneration of other organs such as the kidneys, ovaries, etc. Death usually results from obstruction of the portal vein. When such cystic disease

of the liver is discovered at operation, further operative procedure is contraindicated.

Kehr quotes Pellman as dividing into five classes the non-parasitic cysts of the liver which may be treated by operation:

1. Retention cysts of the biliary tract.
2. Cysts lined with ciliated epithelium, said by von Recklinghausen to be due to retention of mucus.
3. Dermoid cysts.
4. Epithelial cysts, or cystadenomata.
5. Lymph cysts.

The symptoms produced by any of these various classes of cysts are similar to those of the non-infected echinococcus cyst (page 193). These cysts may attain considerable size, and then may be mistaken for cystic conditions of the kidney or ovary. The differental diagnosis is to be made from study of the symptoms presented, together with the anatomical diagnosis of the organ involved. Non-parasitic cysts of the liver cannot be differentiated before operation from echinococcus cyst of the liver, and often it is necessary to depend upon the microscopical examination of the contents or of a section of the cyst-wall for a correct diagnosis. It is not justifiable in any instance to make an exploratory puncture for the purpose of diagnosis.

Treatment.—The treatment consists in the removal of the cyst contents and of as much of the cyst-wall as is possible. In pedunculated cysts, the entire mass usually may be removed. When the cysts are deep seated, an incision through the liver substance must be made, down to the cyst-wall, when it will be found feasible to shell out many of them, without rupture, owing to the fact that the cyst-wall usually is dense. When rupture during removal does occur, there is less danger of serious infection than from a ruptured echinococcus cyst. The postoperative course and treatment are similar to those following the operation for echinococcus cyst. Boyd has collected 34 operations for non-parasitic cysts of the liver, with 11 deaths.

REFERENCES.

Barrier: De la Tumeur Hydatique du Foie, Paris, 1840. (This work has not been accessible to us.)

Boinet: Marseille Méd., 1882, xxix, 803.

Boinet et Chazoulière: Rev. de Méd., 1898, xviii, 845.
Bond: Brit. Med. Jour., 1891, i, 795.
Boyd: Lancet, 1913, April, p. 951.
Bremser: Ueber Lebende Wurmer im Lebenden Menschen. Wien, 1819, S. 244; Les vers Intestinaux de l'Homme, Paris, 1824, p. 293.
Cauchoix: Traitement Chirurgical Actuel des Kystes Hydatique du Foie, et de leurs Complications, Paris, 1908.
Davaine: Traité des Enterozoaires, Paris, 1860. (Only the second edition of this work, published 1877, has been accessible to us.)
Delbet: Bull. de l'Acad. de Méd., Paris, 1896, xxxv, 136, 577.
Dévé: Rev. de Chir., 1907, xxxv, 529, 818, 1013.
Dévé: Thèse de Paris, 1901.
Douglas: Surg. Dis. Abdomen., Phila., 2nd Ed., 1909, p. 317.
Finsen: cited by Johnson: Surgical Diagnosis, N. Y., 1909, ii, 152.
Fowler, R. S.: N. Y. State Journ. of Med., 1906, vi, 275, 322.
Ghedini: Gaz. d. Ospedali, Milano, 1907, xxviii, 53.
Israel: Verhandl. d. Deutsch. Gesellsch. f. Chir., 1879, viii, i, 17.
Kehr: System of Practical Surgery, Ed. by v. Bergmann, Bull, et al., Phila., 1904, iv, 643, 654.
Landau: Berl. klin. Woch., 1880, xvii, 93, 108.
Madelung: Beitr. Mecklenburg. Aerzte z. Lehre von der Echin. Krankh., Stuttgart, 1885. (This work has not been accessible to us.)
Madelung: Mitth. a. d. Grenzgeb. d. Med. u. Chir., 1904, xvii, 21.
Morris: Ashhurst's International Encyclop. of Surgery, Revised Ed., New York, 1888, v, 644.
Pallas: Dissert. Inaug., Berolin., 1760; in Sandifort: Thesaurus Dissertationum, Lugd.-Bat., 1778, Tom. 1, ff. 286–287.
Posadas: Ann. del Circul. Med. Arg., 1895.
Prudden: Delafield and Prudden, Text-book of Pathology, 9th Ed., N. Y., 1911, p. 738.
Quénu: Bull. Soc. Chir. Paris, 1903, xxix, 719. (Formolization).
Quénu and Duval: Bull. et. Mém. Soc. Chir., Paris, 1906, xxxii, 1005 (Rupture into biliary passages).
Richardson: Edinb. Med. Jour., 1867, xiii, 525.
Roser: Giessener Naturforscher-Versamml., 1864; cited in discussion of Israel's paper in Verh. d. Deutsch. Ges. f. Chir., 1879, viii, i, 17.
Russell: Intercol. Quarterly Jour. of Med. and Surg., 1894–95, i, 317.
Ryan: Austral. Med. J., 1895, n. s., xvii, 403.
Ryan: Lancet, 1895, ii, 1080.
Thompson: Brit. Med. Jour., 1896, i, 1332.
Thornton, Knowsley: Med. Times and Gaz., 1883, i, 89.
Végas and Cranwell: Kystes Hydatiques in Republ. Argent., Buenos Aires, 1901 (cited by Cauchoix).

CHAPTER IV.

TUMORS OF LIVER, GALL-BLADDER AND BILE-DUCTS.

Tumors of the liver, apart from those of the gall-bladder and bile-ducts, have little practical interest for the surgeon. They are important, however, from the aspect of diagnosis; but only in a few exceptional cases, so far, has it been possible for the surgeon to attempt any radical cure by means of operation. It is possible that the future will broaden this field of hepatic surgery so that relief may be afforded a greater number of sufferers from hepatic tumors.

Benign Solid Tumors of the Liver.

With the possible exception of adenoma, benign solid tumors of the liver are surgical rarities. **Angeiomata, fibromata, myomata,** etc., have been observed but are principally of pathological interest. As pointed out by Mayo, a diffuse or circumscribed angeiomatous condition of the liver sometimes is found during operations on the bile-passages and troublesome hemorrhage may result if injury occurs to the dilated blood-vessels forming the tumors.

Adenoma of the liver is a comparatively rare neoplastic growth, of very obscure origin. It is not an uncommon postmorten finding, although unfrequently recognized clinically. Gordinier and Sawyer have collected forty-four cases reported by various observers. Cushing and Downs found seven cases of adenomata reported among seventy-five operations for tumor of the liver. Adenomata may occur at any age, cases having been reported in patients twenty months and seventy-six years of age respectively. Of twenty-nine cases, collected by Marckwald, twenty-three were in patients of the male sex.

Pathology.—Adenoma of the liver is a primary growth and two varieties are recognized: the *nodular adenomatous hyperplasia* of the liver, which is not amenable to surgical treatment; and the *circumscribed tubular adenoma* (Langenbuch). Among the forty-

four cases mentioned above, twenty-eight were multiple and sixteen solitary.

In *nodular adenomatous hyperplasia* which usually is associated with cirrhosis of the liver (Simmons) there is a roughened nodular condition of the surface of this organ. Cross-sections of the liver show pathological masses of abnormal color, not definitely encapsulated but separated from each other by fibrous changes in the parenchyma (cirrhosis). Pathologists are not agreed whether the cirrhosis causes the development of these multiple adenomata (as a compensatory hypertrophy), the view held by Rolleston; or whether both pathological changes are due to the same original irritant, as maintained by Engelhardt and Dieulafoy. Brissaud followed Schüppel who held that adenoma is a stage between cirrhosis and carcinoma.

The *tubular adenoma* may be single or multiple, usually the latter. The tumors usually are small, though one may reach the size of a large orange. The larger the tumor the more distinct is its encapsulation. They are grayish-white or yellow in color, unless hemorrhage has occurred into the substance of the tumor, when it has a reddish tinge, or when it has been stained green by bile. The larger the tumor the more apt is internal hemorrhage to occur. *Cystadenoma* may develop in this way or may result from degenerative changes. Metastasis is very rare, but Langenbuch refers to a few reported instances.

Adenoma of the liver may be derived from (*a*) liver cells, (*b*) the intrahepatic bile-ducts, or (*c*) from adrenal rests in the liver (Rolleston). The larger tumors, and particularly those which become cystic, usually are derived from the bile-ducts. Those springing from the hepatic cells sometimes contain tubules lined with cubical epithelium, thus resembling primary carcinoma with cirrhosis. In cases of adenoma of the liver reported by Keen, and by J. B. Roberts, it was found that the adenomatous change was due to proliferation of coccidia.

The **symptoms** of adenomata are chiefly those of cirrhosis of the liver, the symptoms being produced by compression of the vessels and bile-ducts. In the early stages no symptoms may be produced, but as the tumor increases in size digestive disorders arise, accompanied by dull pain in the hepatic region, followed by jaundice and emaciation. Other symptoms, due to

interference with the portal circulation, often develop; varicosities of the abdominal veins may be seen and ascites develops rapidly. In advanced cases a hard, circumscribed mass may be felt, at times pendulous, but moving freely with the liver during respiration. In single tumors, the liver may be of normal size, but in multiple adenoma the liver is enlarged and nodular.

The **prognosis** is bad unless extirpation is possible before the development of obstructive symptoms. But the condition may exist for a long time without giving rise to symptoms of any kind.

The **treatment** is palliative in nodular adenomatous hyperplasia. Even in the case of single adenoma, operative interference is contraindicated after the onset of jaundice and ascites. In the earlier stages, exploratory laparotomy should be done: if the tumor is in a favorable location and easily accessible, it should be removed by partial hepatectomy. The operative mortality is about 15 per cent. (page 215).

Gummata of the liver usually are discussed in connection with hepatic neoplasms; they occur as single or multiple nodules varying in size from a pea to a hen's egg. They are of interest to the surgeon from a diagnostic standpoint, operative interference seldom being adopted except in cases where a wrong diagnosis has been made, or for degeneration or calcification of the gummatous nodules. When first formed the gumma is soft; later a central area of necrosis appears, and if healing is uninterrupted a stellate fibrous cicatrix results, which is quite characteristic and usually easily recognized. Sometimes the gumma becomes calcified. It very rarely undergoes liquefaction necrosis or suppuration from secondary infection. The larger nodules are found principally on the surface of the liver, in the vicinity of the suspensory ligament, although they may occur along the free border or along the upper surface of the right lobe. Occasionally they are pedunculated (Cumston) and may be mistaken for a linguiform lobe (page 158) or a floating liver. Between the gummata may be found cicatricial bands, which penetrate into the liver in the form of deep furrows. When these are present in great numbers a condition known as botryoid liver results, the organ being divided into lobulated masses separated by the cicatricial bundles.

The *symptoms* of gummata of the liver rarely are conspicuous. As a rule the liver is enlarged and this forms the most constant

symptom. Pain may be present on account of the involvement of the serosa. At times a friction rub may be detected. In the cicatricial stages, pain is almost constant, being of a very dragging character, and referred to the hepatic region, sometimes to the right shoulder. In these cases there is almost constant discomfort, exacerbations of pain being noted. Fever is never present unless there is breaking down and ulceration of the gumma. Kirchheim reports three cases of febrile syphilitic lesions in the liver, all three involving the adjoining diaphragm; in one case there was perforation of this structure followed by empyema and chronic inflammation of the lower lobe of the right lung; pleurisy with effusion developed in the second case; in the third case although the diaphragm was involved no thoracic symptoms developed, but the patient died from perforative peritonitis.

Jaundice is rare and occurs only in those cases where there is diffuse syphilitic involvement of the liver.

The *diagnosis* depends on the history of the patient, on the discovery of other syphilitic lesions or the evidence of past lesions; on the presence of the Wassermann reaction; and on the therapuetic test of antisyphilitic remedies. Gummata of the liver must be distinguished especially from disease of the gall-bladder, and from carcinoma. The former usually may be excluded by a careful study of the history. In carcinoma the disease does not extend over so long a time; there is no enlargement of the spleen (common in syphilis of the liver); and the patient very seldom is under forty years of age. At operation the differentiation from carcinoma may be difficult, but carcinoma of the liver almost always is secondary to a primary growth elsewhere; there never are scars of healed lesions on the surface of the liver as is frequently the case in syphilis of the liver; the carcinomatous nodules rarely if ever stand forth prominently from the surface of the liver; and when cut and scraped the carcinoma gives "cancer juice" which is never the case with a gumma.

Treatment.—The usual antisyphilitic measures should be instituted. If the patient's condition is urgent, salvarsan should be administered. In most cases the vigorous use of mercury and the iodides will bring relief of symptoms. If such treatment fails, as it usually does when the gumma is very fibrous or calcified, excision of the portion of the liver affected should be attempted.

Lotheissen summarizes the results of forty such operations; thirty-four patients were cured, two were improved, while four died. These forty cases do not include simple exploratory operations, with separation of adhesions, but only cases of excision.

Tuberculoma of the liver is rare. Lotheissen has collected thirty-four cases, as well as thirteen cases in which there was a tuberculous abscess in or near the liver. In thirty-two instances the lesions in the liver were found only at autopsy; and in twenty-three there had been no symptoms during life to call attention to the liver. Operative treatment was undertaken in fifteen cases, but a correct diagnosis before operation was made in only three. Ten of these patients recovered.

REFERENCES.

Brissaud: Arch. gén. de Méd., 1885, xvi, 129.
Cumston: Annals of Surgery, 1903, i, 726.
Cushing and Downs: Penn'a. Med. Jour. 1898–1900, iii, 306.
Dieulafoy: Manuel de Path. Interne, Paris, 1901, ii, 774.
Douglas: Surg. Diseases of Abdomen, 2d Ed., Phila., 1909, p. 336.
Engelhardt: Deutsch. Arch. f. klin. Med., 1898, lx, 607.
Gordinier and Sawyer: Amer. J. Med. Sc., 1913, i, 258.
Keen: Boston Med. and Surg. Jour., 1893, cxxviii, 237, (adenoma).
Keen: Annals of Surg., 1899, xxx, 267 (gumma).
Kirchheim: Deutsch. med. Woch., 1911, xxxvii, 152.
Langenbuch: Chir. d. Leber u. Gallenblase, Stuttgart 1897, ii, 44.
Lotheissen: Beitr. z. klin. Chir., 1912, lxxxi, 1.
Mayo, W. J. and C. H.: Keen's Surgery, Phila., 1908, iii, 983.
Marckwald: Virchow's Archiv. f. path. Anat., 1896, cxliv, 29.
Roberts, J. B.: Amer. Jour. Med. Sc., 1894, ii, 664.
Rolleston: Dis. of Liver, Gall-bladder and Bile-ducts, Phila., 1905.
Schüppel: v. Ziemssen's Handb. d. spec. Path. u. Ther., Leipzig, 1875, Bd. viii, Hälfte 1, Abth. 1, S. 316.
Stewart, G. D.: Amer. Pract. of Surgery, Ed. by Bryant and Buck, N. Y., 1911, viii, 219.
Simmons: Amer. Jour. Med. Sc., 1877, ii, 463.

Malignant Tumors of the Liver.

Sarcoma of the liver is very rare and clinically cannot be distinguished from carcinoma. In almost all cases it is secondary to sarcoma in some other part of the body, particularly the eye,

and soft tissues of the limbs. There are a few cases on record in which sarcoma of the liver has been secondary to a primary sarcoma of bone, but in none of these was there any osseous tissue in the nodules found in the liver. Langenbuch refers to two instances of secondary chondrosarcoma of the liver. Most tumors are spindle or round celled. Lymphosarcoma, myosarcoma, and myxosarcoma also have been recorded. Melanotic sarcoma is not very rare, especially when secondary to a similar tumor of the eye (choroid).

Knott has collected fifty-nine cases of primary sarcoma of the liver, and reported fifteen hitherto unpublished cases, making a total of seventy-four cases.

The *symptoms* are not characteristic. Pain, enlargement of the liver, perhaps jaundice and ascites, may not develop until late in the disease. Kahlden reported a case in which the first symptom was the black color of the fæces, followed by dark urine, both appearing before there was any demonstrable tumor of the liver. Fever of an intermittent type is not unusual during the course of the disease. In most cases this course is very rapid toward a fatal termination.

The *treatment* usually must consist merely in palliation of the symptoms as they develop. Early exploratory laparotomy may serve to clear up the diagnosis, and in some cases the tumors have been excised with at least temporary benefit to the patients. Knott, in his study of primary hepatic sarcoma, referred to above, found that operation had been done in twenty-seven cases with fifteen deaths, a mortality of 55.5 per cent.

In eighteen cases the tumor was excised; eight patients died, a mortality of 44.4 per cent. In six cases the tumor was inoperable; four patients died, a mortality of 66.6 per cent. In three cases the nature and result of the operation are not recorded.

Of the twelve patients who survived the operation for any length of time, the after history is known in only six cases: three of these patients died in less than four months; one had recurrence after seven months; one died at the end of nine months; and only one (Bardeleben) remained in good health for as long a period as two years.

Carcinoma is relatively the most frequent neoplasm of the liver. It occurs in middle and advanced life and is seen more

frequently in men than in women. It may be either primary or secondary, the latter occurring in about 96 per cent. of the cases. Among 10,000 autopsies, Hale White found ten primary tumors and 240 secondary to carcinoma elsewhere. Secondary carcinoma is of interest from the diagnostic standpoint alone, as it is not amenable to surgical treatment. Primary carcinoma may be removed with success, if the diagnosis is made early enough; but Kehr, in 800 laparotomies for disease in the upper right quadrant of the abdomen, claims that he found only one case where the primary carcinoma was at a stage where cure by radical operation might have been hoped for.

Pathology.—Carcinoma of the liver occurs in three forms:

1. *Massive carcinoma* appears as a whitish or grayish opaque mass, may grow to immense size (5–12 kg.) and usually involves the whole of a lobe, in most cases the right. The growth is well defined from the surrounding hepatic tissue, does not project markedly from the surface, and the general form of the liver is preserved in spite of the enlargement. If a tumor of this kind is found in the liver it is useless to search for a primary growth elsewhere (Langenbuch S. 52) as the hepatic lesion almost surely is the primary tumor.

2. *Infiltrating or diffuse carcinoma* is very rare, and like the massive carcinoma usually is a primary growth. It gives no metastases (Langenbuch) and histologically often is mistaken for portal cirrhosis (page 182) or nodular adenomatous hyperplasia (page 207). The whole liver is affected but its form is little altered except for the increase in all dimensions. Its surface is studded with little smooth round knobs, the size of peas or cherries, and between them the liver tissue is shrunken and retracted. According to some authorities (Fetzer and Perls) this is merely the most advanced form of the massive carcinoma already described.

3. *Nodular or multiple carcinoma* is the usual secondary type and is also the most frequent primary type. Under the latter circumstances most of the nodules are regarded as metastases from one original primary hepatic tumor. The nodules, which are scattered irregularly over the surface of the organ especially at its periphery, are whitish, gray or yellowish masses from the size of a pin-head to that of an orange, though rarely larger than a walnut.

They stand out from the surface of the liver; frequently cause perihepatitis, with resulting adhesions; and when large often become umbilicated as the result of interstitial hemorrhages.

Beadles maintained that a clinical distinction was not difficult between primary and secondary carcinoma, even in this nodular form. *Secondary nodules* are scattered all over the liver, vary in number and size, and may leave little normal hepatic tissue visible, but they are always more or less rounded in form, and are distributed with fair uniformity, not massed particularly about the fossa of the gall-bladder or the quadrate lobe. "*Primary malignant disease* forms a hard, uniform scirrhous mass, if not involving at least close upon the superior wall of the gall-bladder." It spreads backward from this region in the tongue-like prolongations which extend beyond the general mass. This localization is characteristic, and serves to distinguish primary from secondary carcinoma, even when in cases of the former nature there are metastases from the original tumor in other parts of the liver. The main feature by which primary may be differentiated from secondary carcinoma, according to Beadles, is the apparent origin of the former from the region of the cystic duct or neck of the gall-bladder or the liver tissue immediately adjoining, Gallstones were present in eleven out of thirteen cases of primary carcinoma of the liver; but they were found only in two out of sixty-three cases of secondary carcinoma.

Primary carcinoma of the liver is slightly more frequent in men than in women; but in women secondary carcinoma occurs more than twice as frequently as in men chiefly owing to the greater frequency of primary growths in the area drained by the portal vein. More than half the cases occur between the ages of forty and sixty years, but it is not an excessively rare affection even in childhood. In infants and young children, however, the disease usually is primary in the liver.

The *symptoms* of carcinoma of the liver, whether primary or secondary, are not constant. Probably the condition is diagnosed during life in not more than two-thirds of the cases. In cases of secondary carcinoma it is not unusual for the symptoms referable to the liver to overshadow those caused by the primary growth. Anorexia, gradual but progressive failure of strength and loss in weight, and vague digestive disturbances are the most characteris-

tic symptoms; but the diagnosis must be made by exploratory laparotomy unless a palpable tumor has developed.

Cachexia, which develops early, usually advances steadily. This is especially true of the development of secondary growths in the liver, the primary growth from which metastasis has occurred very frequently not causing any cachexia at all.

Jaundice is present in about 50 per cent. of all cases. It varies in degree, especially at first, but usually increases in intensity when present and persists. It may be due to obstruction of the common duct by the pressure of a primary growth in the head of the pancreas; to secondary nodules in the lymph-nodes along the common duct; to primary foci of carcinoma in the biliary ducts; or to direct pressure on the intrahepatic ducts.

Pain may be absent in carcinoma of the liver, but there always is a general feeling of discomfort, a sense of weight and heaviness. The actual pain varies somewhat with the position and extent of the growth. When the neoplasm is deep seated there is not very much actual pain; when the capsule of the liver is involved, the pain may be sharp and cutting in character. With the formation of perihepatic adhesions, pain becomes more constant, often radiating to the epigastrium, the thorax, the back, or the shoulder, and being aggravated by motion. Colicky pain is present at times in those cases where the new growth is near the hilum; it is due to biliary obstruction.

Tenderness may be elicited on deep palpation, but it is not present in all cases.

Ascites is present in about one-half the cases. If jaundice is present the fluid usually is bile stained; otherwise it is clear. Usually it is not excessive in amount, and but little discomfort is experienced from its presence. Tapping seldom is required.

Enlargement of the liver is the most constant phenomenon, the increase in size at times being very great. The right lobe generally enlarges more than the left. The edges may be palpable, firm and hard, but usually are irregular and more or less nodular.

The *diagnosis* often is very difficult to make, unless the cases are well advanced, or unless a primary focus is recognized. In some instances an exploratory operation may be advisable to clear up the diagnosis. The condition must be differentiated especially

from abscess or echinococcus cyst of the liver, syphilis of the liver, and biliary hypertrophic cirrhosis; this usually may be done by careful study of the history and by the physical examination. At times it is impossible to differentiate between carcinoma of the liver and tumors in adjacent structures, such as the stomach, the colon, the kidney or the omentum. In these cases exploratory laparotomy is justifiable.

The *prognosis* is practically hopeless. Hale White gave the duration of life as four months after the development of symptoms in primary carcinoma and seven months after the symptoms of secondary carcinoma began. If a primary growth can be completely removed by excision, the chances of permanent cure are reasonably good; but there are exceedingly few cases in which this treatment is possible. Lücke's patient according to Terrier and Auvray died of recurrence two years after operation (Yeomans gives the period of survival as eight years). Freeman's was in good health over three years after operation; and Schräder's remained in good health even seven years after operation. Hochenegg's patient survived three years, and for two years of this time was in good health.

The *treatment* hitherto has been considered purely palliative. This certainly still is true of secondary carcinoma where operation is contraindicated, but in the future it is more than likely that earlier exploratory operation will reveal an increasing number of cases of primary carcinoma suitable for extirpation. Operation always is contraindicated in the presence of multiple nodules, marked cholæmia, advanced cachexia, and lymphatic enlargements. When the tumor is single, of primary origin, and can be readily reached, partial hepatectomy may be performed. Mayo Robson reports having exposed the liver by operation in thirty-five cases and finding three among them which he considered suitable for hepatectomy. One patient died on the table, and the other two died from recurrence in a few months. Terrier and Auvray collected thirty-eight radical operations for various neoplasms of the liver; thirty-two patients recovered, and six died from the operation, giving an *operative mortality* of 15.8 per cent. The immediate result is known in seventy-four out of seventy-five cases of partial hepatectomy for various causes tabulated by Keen: eleven patients died, an operative mortality of

14.9 per cent. The immediate death rate following operation is largely independent of the nature of the growth. Yeomans collected ten operations for primary carcinoma of the liver, including a case of his own. The following list includes also an operation by Terrier, which Yeomans seems to have overlooked.

(1) Jacobs: Thermocautery and drainage; recovery; recurrence in seven months.
(2) Lapointe (Operation by Segond): Pedicle clamped and divided; death on third day.
(3) Lücke: Elastic ligature and cautery; death two years later from recurrence; (possibly a gumma).
(4) Filippini: Elastic ligature, resection two days later; recurrence and death in two months.
(5) Keen: Thermocautery of entire left lobe; recurrence and death in five months.
(6) Hochenegg: Excised, and stump fixed in abdominal wound; patient died of recurrence three years later; diagnosis doubtful.
(7) L. Freeman: Tumor isolated by gauze strips through liver substance; operation January 20, 1903; excellent health August, 1906.
(8) Schräder: Excised, wound cauterized and fixed in abdominal wound; patient alive and well seven years after operation.
(9) Roux: Excised, bleeding controlled by suture through liver; recovery.
(10) Yeomans: Incised, curetted, packed and drained; well two months after operation.
(11) Terrier: excision; recovered, but three months after operation probably had pelvic recurrence.

REFERENCES.

Bardeleben: Verhandl. d. Deutsch. Gesellsch. f. Chir., 1893, xxii, i, 10.
Beadles: Trans. Path. Soc. London, 1896, xlvii, 69.
Fetzer: quoted by Langenbuch, loc. cit.
Filippini: See Anschütz: Samml. klin. Vortr., 1903, Chir. No. 99.
Freeman: Trans. Amer. Surg. Assoc., 1904, xxii, 196.
Hochenegg: See Anschutz: Samml. klin. Vortr., 1903, Chir. No. 99.
Kahlden: Beitr. z. path. Anat., 1897, xxi, 264.
Keen: Boston. Med. and Surg. Jour., 1892, cxxvi, 405, (Cases 1–20).
Keen: Penna. Med. J., 1897, i, 193 (Cases 21–59).
Keen: Annals of Surgery, 1899, xxx, 267 (Cases 60–75).
Kehr: System of Practical Surgery (v. Bergmann and Bull) Phila., 1904, iv, 656.
Kelly: Osler's Modern Medicine, Phila., 1908, v, 850.
Knott: Surg., Gynec. and Obstet., 1910, vii, 328.
Jacobs: Arch. de Tocol. et Gyn., 1891, xviii, 742.

Langenbuch: Chir. d. Leber. u. Gallenblase, Stuttgart, 1897, ii, 38.
Lapointe: Bull. Soc. Anat. Paris, 1896, lxxi, 700.
Lücke: Zentr. f. Chir., 1891, xviii, 115.
Perls: quoted by Langenbuch, loc. cit.
Schräder: Deutsch. med. Woch., 1897, xxiii, 173.
Terrier and Auvray: Rev. de Chir., 1898, xviii, 403.
White, W. Hale: System of Med. (Allbutt), London, 1908, iv, Part 1, p. 204.
Yeomans: Jour. Amer. Med. Assoc., 1909, i, 1741.
Roux: Rev. Méd. de la Suisse Romande, 1897, xvii, 114.

Tumors of the Gall-bladder and Bile-ducts.

Cystic Degeneration of the Gall-bladder.—This is a rare condition which has been studied recently by Konjetzni. Licini has published another case, in which the gall-bladder was converted into a multicystic tumor the size of an average apple; there was also an adenocarcinoma of the cystic duct; and the pressure of the enlarged gall-bladder on the common duct caused jaundice to develop.

According to Aschoff and Bacmeister there is in about 3 per cent. of gall-bladders an adenomatous structure, especially at the fundus; and in cases of infection which closes these glands, cystic degeneration may occur. They believe it predisposes to the development of carcinoma.

The proper *treatment* is cholecystectomy.

Carcinoma of the gall-bladder and bile-ducts is much more common than carcinoma of the liver. Secondary carcinoma occurs but is of no surgical interest as the lesions rarely develop except late in a general carcinomatosis.

Primary carcinoma of the gall-bladder and bile-ducts is much more common than formerly suspected. It is found in about 5 per cent. of all cases of carcinoma. It occurs three to four times as often in the gall-bladder as in the bile-ducts. Musser, in 1889, was able to collect 100 cases, in sixty-four of which the variety of the new growth was clearly indicated. Among 3908 operations on the gall-bladder and biliary passages, performed between 1890 and 1910, W. J. Mayo found 85 cases or 2.1 per cent. of malignancy. The senior author has found 2.3 per cent. of malignancy in 262 operations performed by him at the German Hospital. It is probable that this proportion of carcinomas in gall-bladder and

biliary duct surgery would be considerably increased if a careful microscopical study were made of every gall-bladder removed and if many of the cases that are too far advanced for operative interference were included in the statistics.

Primary carcinoma of the gall-bladder is most frequently seen between the ages of fifty and sixty; but Pröscher reported a case in a man twenty-two years of age. It is much more frequent in women than in men, the proportion being 3–1 according to Musser, and 4–1 according to Fütterer. Among seven patients operated upon by the senior author, six were women averaging in age from thirty-six to sixty-three; the seventh patient was a male, twenty-six years old.

Schröder says that 14 per cent. of all gall-stone patients eventually suffer from carcinoma of the biliary apparatus. The theory that the irritation of the gall-stones predisposes to the development of carcinoma in the gall-bladder is well borne out by statistics: thus Musser found gall-stones associated with carcinoma in 69 per cent. of the cases; Fütterer, in 78 per cent.; Winton, in 81 per cent.; Zenker, in 91 per cent.; Courvoisier, in 91 per cent.; Siegert, in 95 per cent.; and Janowski, in 100 per cent. In the senior author's series, gall-stones were present in 87 per cent. The theory of irritation as a causative factor in producing carcinoma of the gall-bladder is enhanced by the fact that Beadles, in a study of twenty-eight personal cases of secondary carcinoma of the liver and gall-bladder, did not find gall-stones in a single instance; while in a total of sixty-three cases of secondary carcinoma gall-stones were present in only two instances.

Pathology.—Primary carcinoma of the gall-bladder is most frequently found in the fundus, the secondary site of preference being near the neck of the bladder or the beginning of the cystic duct. In other cases the entire organ may be involved. In his analysis of forty-five cases, Fütterer found the growth in the fundus in seventeen, near the opening of the cystic duct in thirteen, on the posterior wall in eight, and on the anterior wall in seven.

Most cases are of the columnar-cell type; but Konjetzni has collected twenty-three instances of squamous-cell carcinoma of the gall-bladder.

There are two types of new growth recognized, although in any case they may coexist. In one there is a cauliflower-like growth which projects into the cavity of the gall-bladder; in the second form there is a general infiltration of the walls of the organ. Although the growths usually are circumscribed in the beginning, infiltration becomes marked and nodular enlargement of the gall-bladder results. If the cystic duct is obstructed, the gall-bladder usually becomes distended; the contents usually are bloody. Perforation into the general peritoneal cavity rarely ensues, although adhesions to the surrounding viscera are common; in this way stenosis of the duodenum, pylorus, or colon, may occur. In some cases ulceration into the colon, stomach or duodenum with the formation of an internal biliary fistula may result. An external biliary fistula is very rare, except as the result of operation. Extension to the liver is found in about 50 per cent. of the cases, the process in the liver being the result of direct extension along the biliary ducts or by the lymphatics. As noted at page 213, Beadles inclines to the view that most if not all cases of primary carcinoma of the liver arise at or near the cystic duct or neck of the gall-bladder.

The following case history shows the possibility of inflammation and irritation other than that caused by calculi acting as an ætiological factor in gall-bladder carcinoma.

A. F., female, forty-nine years of age was admitted to the German Hospital in January, 1907.

Family history, negative. No tuberculosis or malignancy.

Previous History.—Has had seven children. Thirteen years ago had an attack of acute nephritis; four years ago an attack of pneumonia; one year ago an attack of acute appendicitis at which time there was marked pain in the upper right quadrant of the abdomen, and patient was jaundiced. *Chief complaint on admission* to hospital was profuse, foul-smelling, at times bloody, vaginal discharge.

Physical examination: large, stout, well-nourished woman, with flabby, pendulous abdomen. No pain or tenderness over entire abdomen. Vaginal examination revealed presence of a small, freely movable uterus with normal appendages. A small polyp was protruding from cervix. Two days after admission patient was suddenly seized with pain over gall-bladder region followed by rise of temperature, marked tenderness and rigidity over the gall-bladder and slight jaundice. Patient rapidly recovered from the gall-bladder attack.

Operation: March 30, 1907.—The uterus was curetted. Pathological

examination showed absence of any malignancy. Upper right rectus incision made. Adhesions of some standing between the gall-bladder and stomach and duodenum; new adhesions between gall-bladder and liver. Adhesions ligated and cut and gall-bladder freed. Gall-bladder aspirated and reddish-brown bile removed. Gall-bladder opened. No gall-stones found. Cystic and common ducts patulous. Mucous membrane swollen and congested. Cholecystostomy with protective gauze and rubber-dam drainage.

Subsequent Course.—Patient made a good recovery. Was relieved of all trouble until the summer of 1908, when she complained of a dull, aching pain about the incision. Pain was more or less constant, with sharp exacerbations. Has been nauseated with vomiting. During and after very sharp pains would become jaundiced. Readmitted to the German Hospital, July 17, 1909.

No jaundice. Great rolls of fat on pendulous abdomen. Sense of a mass near old scar. Tenderness on palpation.

Second Operation, July, 1909.—Upper right rectus incision. Gall-bladder region found to be a dense mass of tissue, evidently carcinoma. Numerous carcinomatous nodules on lesser omentum and about gall-bladder site. Nodule removed. Wound drained and closed to drainage. Pathological report of nodule proved it to be carcinoma.

Patient made an operative recovery and was discharged from the Hospital August 6, 1909. Subsequent history not known.

Symptoms.—These are much the same as those of primary carcinoma of the liver, especially when the tumor arises in the mucous membrane of the gall-bladder; the carcinoma that arises from the glands of the gall-bladder's mucous membrane is more apt to produce symptoms referable to the gall-bladder itself or its ducts. In the vast majority of cases the patient who presents a hard nodular tumor of the gall-bladder or liver which may be diagnosticated certainly as carcinoma, without an exploratory operation, is already beyond the help of surgery.

Treatment.—The most favorable cases are those where a thick-walled gall-bladder removed at operation is recognizably affected by carcinoma only after microscopical study. Most cases recognized as malignant during the operation prove too far advanced for excision to be justifiable; or if excicion is done, the patients die of recurrence within a year. On the other hand, among seven cases recognized as carcinomatous only after microscopical study, Mayo had three patients who were well more than two years after operation. But whenever possible, even in cases recognized as malignant, excision of the gall-bladder, cystic duct

and of the adherent surface of the liver, should be done. Palliative operation is of little use in relieving the most distressing symptoms, and in most cases merely entails upon the patient the additional discomfort of a biliary or mucous fistula. If a radical operation cannot be done, the abdomen should be closed without doing anything else. Terrier and Auvray collected sixteen cholecystectomies for carcinoma of the gall-bladder, recognized as such at operation; there were five deaths (an immediate mortality of 31 per cent.) and eleven rapid recurrences. Among eighteen operations where a portion of the liver was removed along with the gall-bladder, there were three immediate deaths (16.6 per cent.) and fourteen deaths from recurrence in from six to eight months. One patient (that of Hochenegg, already referred to at page 216) survived for three years, during two years of which time he enjoyed good health. These more radical operations had all been done since 1890. Terrier and Auvray came to the pessimistic conclusion that while the end results of radical operation for this condition were detestable, yet those of palliative operation were still more detestable. They found among fifteen cholecystostomies in cases of malignant disease, that four patients died from the operation, and only one survived as long as a year. Palliative operation should be done only for signs of grave cholecystitis or cholangeitis.

Primary Carcinoma of the Bile-ducts.—This occurs in men, in about 61 per cent. of cases; thus showing a marked contrast to carcinoma of the gall-bladder, which occurs in men in only about 20 or 25 per cent. of cases. In an analysis of sixty cases of carcinoma of the biliary ducts, Schüller found forty-one at the papilla of Vater, and nineteen in the common duct or the hepaticus. Rolleston found the growth situated as follows in eighty cases: In the common bile-duct, thirty-three (lower end twenty-one, mid-part, eleven); at the junction of the common bile-duct, cystic duct and common hepatic duct, twenty-five; in the common hepatic duct, eighteen; in the right or left hepatic ducts, three; in the cystic duct, one; and in the cystic duct and lower end of the bile-duct, one (Kelly). In 102 cases, Donati found twenty-nine of the choledochus, thirty-four at the hepatico-cystic juncture, twenty-eight of the hepaticus, one of the cysticus, and ten not located. According to Licini his case of carcinoma of the cystic

duct is the third on record. The result of the growth of carcinoma of the ducts usually is a constriction, with dilatation of the ducts above the growth. The obstructing tumor occurs either as an annular growth which concentrically constricts the duct, or as a papillary outgrowth into the duct lumen. Wide infiltration of the ducts is rare. Carcinoma at the papilla of Vater may be of *intestinal*, *pancreatic*, or *biliary* origin. Clinically it resembles cylindrical-celled carcinoma of other portions of the intestinal tract in its slow and superficial growth, and its slight tendency to metastasis (Terrier and Auvray). Obstruction in the common duct results in distention of the gall-bladder unless this has been previously diseased and contracted. When the growth is in the hepatic duct, the gall-bladder usually is small. Enlargement of the liver occurs in almost all cases, and there may also be associated obstruction of the portal circulation and the development of ascites.

Symptoms.—In carcinoma of the extra-hepatic biliary ducts the symptoms are not marked, as a rule, until partial obstruction of the duct occurs. As in all conditions affecting the biliary tract, symptoms of dyspepsia may be noted, but usually the first symptom that calls attention to the biliary apparatus is the onset of jaundice. In most cases the jaundice develops gradually, although rare cases may show sudden icterus similar to calculous obstruction. The jaundice of carcinoma of the duct is permanent and never intermittent.

Pain, of a dull aching character, may be noted in the gall-bladder region or in the epigastrium. When a distended gall-bladder attempts to empty itself, there may be colicky pains simulating gall-stone colic.

The gall-bladder usually is enlarged on account of the obstruction below, and is palpable in more than 50 per cent. of the cases. The liver is slightly enlarged.

The *diagnosis* must be made from calculous obstruction of the common duct. In the latter cases, the *jaundice* develops suddenly, is intense, but intermittent, the stools are not constantly acholic; in carcinoma it develops slowly, steadily increases and never becomes intermittent, no bile being found in the fæces at any time after complete obstruction has once developed. In calculous obstruction, the *pain* is sudden, severe, radiates to the right shoulder, and is accompanied by marked tenderness and rigidity in the re-

gion of the gall-bladder; in carcinoma there is only a dragging sensation, often attributed to gastric disorder. In calculous obstruction the *temperature* usually is elevated, and the attack may be ushered in by a chill; in carcinoma there rarely is fever. In the former case the patient's *general nutrition* may be preserved for a long time, and the patient's appetite and digestion depend on the intensity of the colic and jaundice; in carcinoma, on the other hand, strength is quickly lost, signs of duodenal obstruction supervene, and emaciation is rapid.

An enlarged gall-bladder, in the presence of increasing and non-remitting jaundice, is due to carcinoma in a large majority of cases; the jaundice due to common duct obstruction is accompanied by a contracted gall-bladder in 84 per cent. of the cases, according to Courvoisier. Before enlargement of the gall-bladder occurs diagnosis is very difficult; but this enlargement develops so constantly as soon as obstruction is complete, that there is little excuse for failure to recognize the true lesion after the development of jaundice. Disturbance of the pancreatic functions indicates a growth at the papilla of Vater or a carcinoma of the pancreas (page 363); distinction is difficult.

Prognosis.—The disease is fatal unless all of the diseased structures can be removed by operative means. The ultimate outcome of the condition is greatly modified by the form of treatment and by the stage of the growth when operation is performed. If the diseased structures are removed early, the prognosis is fairly favorable; if the growth is not entirely removed, or if the disease is so far advanced as to be inoperable, death usualy results in from six to eight months.

Treatment.—Prophylatic treatment should be instituted in all instances by removal of all gall-stones that may be in the gall-bladder and ducts as soon as their presence has been determined. The treatment of carcinoma of the *common duct* is either palliative or radical, consisting in the formation of a fistula between the gall-bladder and the duodenum (cholecysto-duodenostomy) or in a resection of the duct with removal of the growth. Resection of the duct and the various steps necessary to restore a channel for the discharge of bile into the intestinal tract have been discussed at page 124.

Removal of a growth from the **papilla of Vater** or from the **duo-**

denal end of the common duct may be accomplished through a transduodenal incision, as first employed by Czerny in 1901 (Schüller). Oppenheimer has collected eighteen operations of this kind, as shown in the subjoined table; fifteen of the operations were for carnoma with six deaths; one for benign tumor, and two for cicatricial stricture, all three successful.

TRANSDUODENAL EXCISION OF PAPILLA OF VATER.

(Oppenheimer's Table.)

Operator.	Lesion.	Result.
Cordua	Carcinoma	Recovered
Cuneo	Carcinoma	Died, 2 days
Czerny	Carcinoma	Died, 5 days
Hotz	Carcinoma	Recovered
Körte	Carcinoma	Recovered
Körte	Carcinoma	Died, 9 days
Körte	Benign cicatricial stricture	Recovered
Kraske	Carcinoma	Recovered
Mayo	Carcinoma	Recovered but recurrence in 18 months.
Mayo	Carcinoma	Died, 8 weeks
Morian	Carcinoma	Recovered and well 9 months
Navarro	Carcinoma	Recovered and well 2 years
Oehler	Benign cicatricial stricture	Recovered
Quénu	Carcinoma	Recovered
Stein	Carcinoma	Recovered
Tédenat	Papilloma	Recovered
Verhoogen	Carcinoma	Died, 13 days
Völcker	Carcinoma	Died, 2 days

18 operations, 12 recovered, 6 died, mortality 33 per cent.

Upcott has since recorded a successful transduodenal excision of a carcinoma of the papilla Vater; the patient was doing well one month after operation. Outerbridge, in his careful study of this subject, notes also a recent operation (unsuccessful) recorded by Slajner.

In cases where the growth was too extensive for transduodenal excision, a few attempts at more radical excision have been made (Halsted, Körte, Kausch). The operation in these cases was similar to the cephalic pancreatectomy of Sauvé (page 477). Halsted's patient survived for six months, but Körte's patient died in three days. Kausch advises the following technique, which he has employed successfully, though his patient died nine months later of cholangeitis.

I. Primary operation. Cholecystenterostomy, as a temporary means to restore the patient's health. It is worth noting, however, that according to Quénu's figures (quoted below) seventeen out of twenty-one patients treated by this operation did not survive.

II. Extirpation of the growth.

1. Mobilization of the duodenum.
2. Posterior gastro-jejunostomy.
3. Section and closure of pylorus.
4. Enucleation of duodenum and adjacent pancreas.
5. Section of choledochus and pancreatic duct.
6. Duodenum is sectioned below the growth, and its distal segment is sutured over remains of pancreas, the intervening segment with the stump of the common duct and the pancreas being removed.
7. Choledocho-enterostomy.

The second stage of Kausch's operation, as described above, required four hours for its completion. In cases where the carcinoma is in the **retroduodenal portion of the choledochus,** excision may be attempted after mobilization of the duodenum. Oppenheimer has collected eighteen such operations, ten of which were for carcinoma, with six deaths; three for calculous obstruction, with no deaths; four for benign cicatricial stricture, with two deaths; and one fatal operation for benign tumor. These figures may be seen in the annexed table.

RETRODUODENAL RESECTION OF CHOLEDOCHUS.

(Oppenheimer's Statistics.)

Operator.	Lesion.	Result.
Bier	Carcinoma	Died, 7 days
Doberauer	Carcinoma	Died, 3 months
Enderlen	Carcinoma	Recovered; well 1 year later
Jaboulay	Carcinoma	Died, 4 days
Jenckel	Benign cicatricial stricture	Recovered; well 8 weeks later
Kehr	Carcinoma	Recovered; died 2 1/2 years of hepatic abscess
Kehr	Calculus	Recovered; well 3 months later
Kehr	Calculus	Recovered; well 6 weeks later
Kehr	Benign cicatricial stricture	Died, 3 months (3 days after another operation for recurrent stricture)
Körte	Benign cicatricial stricture	Died, 13 days

RETRODUODENAL RESECTION OF CHOLEDOCHUS.—(*Continued.*)

(Oppenheimer's Statistics.)

Operator	Lesion	Result
Littlewood	Carcinoma	Recovered
Mayo	Carcinoma	Recovered; died 13 months of recurrence
Mayo	Carcinoma	Died, 3 days
Mayo	Carcinoma	Died, 9 weeks
Mayo	Benign cicatricial stricture	Recovered; well 10 months later
Robson	Carcinoma	Died, few days
Rotter	Benign tumor	Died, 3 days
Verhoogen	Calculus	Recovered; well 1 month later

18 operations, 9 recovered, 9 died, mortality 50 per cent.

After excision of the growth, the continuity of the bile-tract must be restored as already described (page 124).

Operations in the presence of obstructive jaundice from carcinoma are very serious undertakings on account of the great danger from hemorrhage. Quénu mentions eight cases of simple exploratory laparotomy: seven of the patients are known to have died of collapse or hemorrhage in a short time. In thirty-six cases in which the gall-bladder was drained, nine survived for periods varying from a few weeks to six months; the others all died from hemorrhage before the eighteenth day.[1]

Of seven cases in which hepaticus drainage was done, only one patient survived more than a few weeks.

Of twenty-eight anastomotic operations collected by Quénu, for carcinoma of the bile-ducts, there were

21 cholecystenterostomies, with 17 deaths, 4 recoveries.
3 choledocho-enterostomies, with 2 deaths, 1 recovery.
1 cystico-duodenostomy, 1 recovery.
2 hepatico-gastrostomy or enterostomy, 1 death, 1 recovery.

From these figures it is very evident that surgeons are correct in their present attitude toward cholecystostomy and cholecystenterostomy for malignant disease: we believe it is generally recognized that these operations should not be performed in such

[1] Quénu recommends, and has employed since 1907, the prophylactic use of antidiphtheritic serum, which is the most easily procured alien serum. He injects 20 c.c. of this on the day before the operation, and since using this precaution has had only one case of postoperative hemorrhage in these icteric patients. Other measures employed as prophylaxis against bleeding have been discussed at page 31.

cases, and that unless a radical operation can be performed the surgeon should content himself with exploration, which, according to Quénu's figures is quite serious enough.

Sarcoma of the gall-bladder and biliary ducts has been described and a few cases are on record. It is impossible to differentiate between sarcoma and carcinoma, the symptoms, diagnosis, prognosis and treatment being practically the same.

REFERENCES.

Beadles: Lancet, 1895, ii, 1168; Trans. Pathol. Soc., London, 1896, xlvii, 69.
Courvoisier: Path. u. Chir. d. Gallenwegen, Leipzig, 1890.
Donati: La Clin. Chir., 1909, quoted by Licini, loc. cit.
Fütterer: Ueber die Aetiologie des Carcinoms, Weisbaden, 1901, S. 55.
Halsted: Bost. M. and S. J., 1899, cxli, 645.
Janowski: quoted by Kelly, l. c.
Kausch: Verh. d. Deut. Ges. f. Chir., 1911, xl, 292.
Kausch: Zentralbl. f. Chir. 1909, xxxvi, 1350; 1911, xxxviii, 100.
Kelly: Osler's Modern Med., Phila., 1908, v, 850.
Konjetzni: Anat. Path. Mitt., 1911, quoted by Licini, loc. cit.
Körte: Beitr. z. Chir. der Gallenwegen u. d. Leber, Berlin, 1905, S. 223, 341.
Licini: Beitr. z. klin. Chir., 1911, lxxvi, 770.
Mayo: Keen's Surgery, Phila., 1908, vol. iii, p. 1005.
Mayo: Collected Papers, Phila., 1910, p. 144.
Mayo: Collected Papers, previous to 1909, Phila., 1912, i, 404 (carcinoma of papilla of Vater).
Musser: Trans. Assoc. Amer. Phys., 1889, iv, 302.
McBurney: N. Y. Med. Jour., 1891, liii, 520; Annals of Surg., 1898, xxviii, 481, Case 2.
Oppenheimer: Deutsch. Zeit. f. Chir., 1912, cxv, 415.
Outerbridge: Ann. Surg., 1913, i, 402.
Prŏscher: Jour. Amer. Med. Assoc., 1907. i, 480.
Quénu: Rev. de Chir., 1909, xxxix, 462.
Schröder: Dissert. Inaugural, Strassburg, 1892. (This work has not been accessible to us.)
Schüller: Beitr. z. klin. Chir., 1901, xxxi, 683.
Siegert: quoted by Kelly, l. c.
Terrier and Auvray: Chir. du Foie et des Voies Bil., Paris, 1901, i, 256.
Upcott: Annals of Surgery, 1912, ii, 710.
Winton: quoted by Kelly, l. c.
Zenker: quoted by Kelly, l. c.

In addition to the patient whose history is detailed at page 219, the following case histories of patients with malignant disease

of the gall-bladder or liver who have been under the senior author's care at the German Hospital may prove of interest:

Carcinoma of Gall-bladder and Pancreas; Cholecysto-duodenostomy; Death.

M. H., female aged fifty-one, admitted to the German Hospital April 27, 1903. Complained of pains in lower abdomen referred to back. These were cramp-like in character. Had nausea and vomiting. Jaundice present but no itching. Night sweats. Stools clay-colored. Liver enlarged and below umbilicus. Marked tenderness over liver and gall-bladder. Systolic heart murmur at apex. Heart weak and irregular. Patient very weak. Hæmoglobin 75 per cent., R. B. C., 4,649,000; W. B. C., 14,000. Coagulation time, four and one-half minutes.

Operation.—Ether anæsthesia. Incision through upper right rectus. Adhesions surrounding gall-bladder, which was enlarged, elongated and thickened. Portion resected. Six large gall-stones in gall-bladder. Pancreas enlarged and evidently obstructing common duct. Cholecysto-duodenostomy performed with Murphy button. Patient did not react from shock of operation.

Autopsy revealed mesenteric and retroperitoneal lymph nodes, enlarged. Peri-pancreatic growth obstructing common duct. Liver much enlarged and bile stained.

Pathological report of gall-bladder: Carcinoma with necrosis.

Carcinoma of Gall-bladder; Cholecystectomy; Drainage of Cystic Duct. Recovery.

R. H., twenty-six years old, single, admitted to the German Hospital March 28, 1907, discharged April 13, 1907.

Family History.—Parents, one brother and three sisters living and well. No history of tuberculosis or malignancy.

Social History.—Single. Farmer for two years. Worked in city before that. Habits regular. Smokes. Uses alcoholics moderately and not constantly. Denies venereal history.

Previous Medical History.—Had measles in childhood. No serious illness since then until present trouble began.

Present Illness.—In September, 1906, just after the evening meal one day, patient was taken suddenly with a crampy pain in the gall-bladder region. Shortly afterward he vomited and felt considerably relieved. Was quite sick, however, for two or three hours. Felt well the next day and went about his work. Was not jaundiced and had no typical attack of biliary colic. Since this primary attack, there have been numerous similar ones lasting, for the most part, only a day or two. It was not until the third or fourth attack that the patient became jaundiced and thereafter there was jaundice with each attack. The skin always cleared up between the attacks.

About four or five weeks before admission patient had the latest seizure and on this occasion was confined to bed for some weeks. Was jaundiced and had a number of attacks of cramp in the epigastrium. Never had any typical attack with chills, sweats and fever. However, he had a profuse sweat when the pain became severe. Always vomited with the attacks. States that in January of this year he passed "gall-stones" in his stool. No other subjective symptoms except that the patient has lost about 27 pounds in weight since the first attack and has lost strength also.

Physical Examination.—Patient is fairly well developed; not so well nourished. Has an anæmic appearance and the skin of the abdomen has a slight yellowish hue. Conjunctivæ are icteroid. Pupils negative. Tongue coated with a yellowish fur posteriorly.

Chest: Lungs and heart in good condition.

Abdomen: Soft, almost scaphoid. Probably a slight rigidity over gall-bladder region. The gall-bladder doubtfully palpable. Kidneys not palpable. Liver and spleen normal to percussion.

Extremities negative.

Operation.—March 30, 1907. Dr. Deaver. Ether. Right rectus incision about 12 cm. long, starting at ninth costal cartilage. Peritoneal cavity opened: gall-bladder palpated and found to be enlarged and tense. Apparently filled with calculi. Adhesions between gall-bladder and greater omentum discovered. These adhesions were freed and the gall-bladder brought up into the wound. Several gauze pads used to protect the peritoneal cavity. At the fundus of the gall-bladder and near the neck several dark objects appearing like calculi but not so hard to the touch, projected through the deeper layers of the wall of the gall-bladder and seemed to be covered only by the serous coat. The gall-bladder was tense and considerably larger than normal as though it were overfilled with calculi. Extirpation of the organ was deemed advisable. Serous covering of cystic duct incised and duct and vessels clamped separately and cut through, and gall-bladder dissected free from its bed. Several bleeding points caught and ligated. Vessels of the gall-bladder ligated with iodine gut. Clamp removed from cystic duct, and duct drained with rubber tube. Cigarette drain placed below cystic duct. One piece of plain gauze put in bed of gall-bladder to control hemorrhage. Duodenum, stomach and pancreas examined; apparently normal. Gauze pads removed. Drainage brought out through a counter-opening. Abdominal wound closed in layers. A few through and through silkworm-gut sutures employed. Stomach washed out third and fourth days after operation; greenish fluid evacuated. After the drainage was removed, wound separated in upper end and required packing. Patient insisted on going home April 13, 1907.

Urine.—Practically negative findings.

Blood.—Hb., 76 per cent., W. B. C., 9500 and coagulation time, one and one-half minutes.

Stomach Contents.—Free HCl, 27; total acidity, 47; Lactic acid, occult blood and microscopical findings negative.

Stool.—Formed stool light brown color. Bile and bile-salts negative. Occult blood negative. Microscopically negative.

Culture.—From gall-bladder sterile.

Pathological Diagnosis of Gall-bladder.—Carcinoma.

Appendix.—Inconspicuous lesions.

CARCINOMA OF GALL-BLADDER; CHOLECYSTECTOMY; RECURRENCE.

M. S., 53 years old, married 27 years, was admitted to the German Hospital December 16, 1906.

Chief Complaint.—Feeling of distress in gall-bladder region.

Family History.—Father, mother, three sisters and one brother living and well. One sister died of pulmonary tuberculosis. Several brothers and sisters died in infancy. No malignancy.

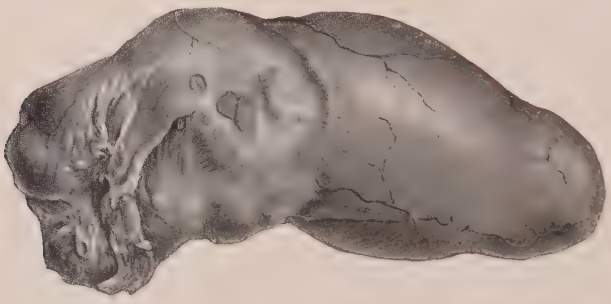

FIG. 17.—GALL-BLADDER REMOVED BY CHOLECYSTECTOMY. FOUND ON MICROSCOPICAL EXAMINATION TO BE CARCINOMATOUS. (*Case of M. S., page 230.*)

Social History.—Two children living and well. Youngest is sixteen years old. Had one miscarriage between two living children. Forceps delivery for both children. One child born dead. One died of diphtheria eighteen years ago. Patient passed menopause two years ago, and has no vaginal discharge.

Previous Medical History.—Measles in childhood; otherwise negative. Appetite good and bowels regular before present illness.

Present Illness.—Has slowly developed a feeling of distress in the gall-bladder region with loss of appetite, weight and ambition. Gradually lost about fifteen pounds. No acute attacks of pain. Never vomited blood. Jaundice of gradual onset.

Operation.—December 18, 1906, by Dr. Deaver. Right rectus incision. Liver raised, and gall-bladder exposed. Field of operation walled off with gauze. The gall-bladder was surrounded almost completely by omental adhesions, which could be stripped off easily with gauze. The gall-bladder was enlarged, distended and full of stones of various sizes. The neck of the gall-bladder just above the cystic duct was folded so as to form a small sac pressing upon the duct. Cholecystectomy decided upon: gastro-hepatic omentum grasped and opened; cystic duct clamped and cut, and cystic vessels

clamped and cut. Gall-bladder pulled free from liver and removed. Cystic duct tied with chromic catgut, and cystic vessels with linen thread. One cigarette drain placed about cystic duct, and sewed fast with chromic gut. Several bleeding points in bed of gall-bladder clamped and tied with catgut. A piece of plain gauze rolled and placed in bed of gall-bladder to control hemorrhage, and held in place by catgut stitch. Pads and sponges removed. Viscera replaced. Wound closed in layers.

Progress.—Persistent jaundice after operation, relieved greatly after removal of second piece of gauze (Dec. 31, 1906), which had to be pulled very hard.

Discharged from the hospital, January 26, 1907.

Re-admitted to the German Hospital, April 1, 1907, less than four months after operation. Has continued to complain of weakness since leaving the hospital. Went home in a carriage and has been in bed ever since. Has had pain in the right side, with nausea and vomiting. A few weeks ago she could not keep anything on her stomach. Has had sweats with remittent and intermittent temperature. Has lost weight, but does not know how much. Has lived on liquid diet ever since leaving the hospital.

Physical Examination.—"Lemon colored" complexion. No distinct jaundice. Heart and lungs negative.

Abdomen: Old scar and stab-wound from gall-bladder operation. Line of induration readily felt along line of drainage. Lower border of liver palpable down almost to the umbilicus. Upper border of absolute dullness about fifth interspace. Spleen not palpable or demonstrably enlarged.

Progress.—Gradual loss of weight, health and strength. At time of discharge from hospital May 14, 1907, the drainage tract had increased greatly in size and was very hard, adherent to skin.

Urine.—Shows albumin.

Stool.—Free fat, bile and occult blood negative.

Blood.—Hb., 54 per cent., W. B. C., 10,700.

Differential Count.—Polys., 82.5 per cent.; lymph., 13; Trans., 4; Mon., 0; eosin., 0; mast., 0.5.

April 25, 1907: Hb., 45 per cent., W. B. C., 10,900.

Cholelithiasis; Carcinoma of Gall-bladder and Liver. Cholecystectomy and Choledochostomy. Recovery (Operative).

Mrs. S. W., fifty-six years old, married, admitted to the German Hospital May 28, 1908, discharged June 21, 1908.

Family History.—Father died of senility, mother in childbirth. Two sisters living and well. One brother dead—accident. No cancer or consumption nor hæmophilia.

Social History.—In U. S. fourteen years. Married twice: first at age of twenty-two; first husband died in Australia the result of excessive use of alcohol. Married sixteen years to present husband. One child living and

well. Five children dead; all died in infancy except one, who died of enteric fever at eleven years. No children by present husband. Menopause six years ago. Her physician says she had syphilis twenty-five years ago.

Previous Medical History.—Usual children's diseases. Has had acute articular rheumatism.

Present Illness.—About six weeks ago, at night was suddenly seized with a severe attack of pain in upper right abdominal quadrant. Vomited and had a chill. Since that time has had a constant gnawing pain in this area. Pain is referred to the back, beneath angle of right scapula. Two days before admission became slightly jaundiced. Has never been a sufferer from indigestion, but during past six weeks has been careful in regard to her diet for fear that indiscretions would increase her pain. Has lost considerable weight in past eight months. Weighed about 200 pounds at that time. Lately has been troubled with gaseous eructations.

Physical Examination.—Well developed and nourished.

Chest: Lungs clear. Heart sounds of good quality.

Abdomen large, flabby, flaccid.

Liver down to level with umbilicus. Gall-bladder palpable.

Operation.—May 30, 1908. Dr. Deaver. Ether. Incision through upper right rectus. Liver down nearly to level of umbilicus and on its anterior surface was a nodular area very like carcinoma. The omentum was adherent to the gall-bladder which was very much thickened and contained stones. Intestines walled off with gauze pads. Liver delivered and gall-bladder opened through neck and two black faceted stones removed. Incision continued through cystic duct and another stone removed. Probe passed through common duct. Cystic duct and cystic artery clamped and cut and gall-bladder dissected from its fibrous bed. Cystic duct and vessels tied with iodine gut. Rubber tube sutured in common duct with catgut; one split tube containing a piece of gauze placed in bed of gall-bladder and the gauze used to reinforce tube in common duct, and the whole held *in situ* with catgut sutures through liver and bed of gall-bladder. Large rubber tube placed in subhepatic space. Gauze pads removed and wound closed in layers with iodine gut and a few through-and-through sutures of silkworm gut tied over gauze. Left the hospital June 21, 1908 with small wound discharging bile.

Urine.—Negative.

Blood.—Hb., 88 per cent., coagulation time, 55 seconds. W. B. C., 7360.

Stool.—Grayish liquid contaihing a few gray fæcal masses and numerous particles of vegetable residue. Moderate odor. Bile test negative. Occult blood negative. Parasites and calculi none. Considerable granular matter, numerous fat globules. No starch cells. Few epithelial cells. No parasitic ova.

Culture from gall-bladder.—Pyocyaneus.

Pathological Diagnosis.—Mucous membrane granular and hemorrhagic and bile stained.

Microscopically.—Carcinoma of gall-bladder and chronic cholecystitis.

CHOLELITHIASIS; CARCINOMA of GALL-BLADDER and LIVER. CHOLECYSTECTOMY. UNIMPROVED.

Mrs. M. R., sixty-three years old, widow, admitted to the German Hospital June 9, 1908, discharged July 11, 1908.

Family History.—Father dead—accident. Mother dead—senility, ninety-three years. One brother living and well; one brother dead—strangulated hernia. No cancer, consumption or hæmophilia.

Past History.—Has been a widow twelve years. Husband died of paresis. Four children in good health. No miscarriages. Menopause about twelve years ago.

Previous Medical History.—Usual children's diseases and scarlet fever. Diphtheria at twenty years. Never had typhoid fever.

Present Illness.—Began about the middle of March, 1908, with gradual onset of pain, aching in character, and situated beneath right costal margin. Pain was occasionally referred to back. The past thirty-five years has had attacks of severe colicky pain in epigastrium accompanied by nausea and vomiting which often lasted several days. Pain was relieved only by inhalations of choloroform. These attacks reached their maximum of intensity about ten years ago. Has never been jaundiced. Never observed anything abnormal in character of stools. For past few years only has she been at all careful in selection of her diet. Says that no variety of food has any influence in provoking an attack of pain, and formerly ate anything she wished between the paroxysms.

Physical Examination.—Well developed and nourished. Quite stout.

Chest: Lungs clear. Heart sounds distant and very little difference in character of first and second sounds. No murmurs.

Abdomen: Large, fatty. Rather tender mass, palpable about in nipple line.

Operation.—June 13, 1908. Dr. Deaver. Ether. Abdomen opened by straight incision through upper right rectus. Intestines packed off with gauze. Gall-bladder enlarged, distended and adherent to omentum and on under surface to pylorus. Adhesions separated. Neck of gall-bladder opened and a large stone size of a walnut removed. Gall-bladder clamped and cut at neck and then dissected downward from its fibrous bed. On being opened it was found to contain pus and a number of gall-stones. The walls were greatly thickened and indurated and apparently the seat of carcinomatous infiltration. The remaining portion of neck of gall-bladder was now removed. Four clamps left on bleeding points and cystic duct; common duct probed. A carcinomatous nodule was found on under surface of right lobe of liver. A piece of gauze was placed in subhepatic fossa, about clamps and a cigarette drain was placed in bed of gall-bladder and held *in situ* with catgut sutures. Gauze pads removed and wound closed in layers with iodine gut. Several through-and-through sutures tied over gauze.

July 11, 1908. Patient removed from hospital in a very weak condition and with a discharging biliary sinus.

Urine.—Very faint trace of albumin. Few granular casts.

Blood.—Hb., 73 per cent.; W. B. C., 7850; coagulation time, thirty-five seconds.

Culture from gall-bladder sterile.

Pathological Diagnosis.—Carcinoma of gall-bladder.

Cholelithiasis and Carcinoma of the Liver. Cholecystotomy. Improved.

E. W., thirty-six years old, single, admitted March 10, 1909, discharged March 27, 1909.

Family History.—Negative.

Menstrual History.—Began at fourteen years, always regular. Last period one week ago.

Past History.—Diphtheria and scarlet fever in childhood.

Present Illness.—Present trouble dates back about one and one-half years when patient had two attacks of sharp pain in right hypochondrium lasting only a few minutes. No vomiting. No further attacks for several months; then began to have rather frequent attacks of longer duration, which seemed to be brought on by eating. For last month has had constant rather severe pain in right hypochondrium radiating to back. Pain now is not influenced by food. No vomiting. Never jaundiced. No chills or fever.

Physical Examination.—Rather fat woman. Heart and lungs negative.

Abdomen: Soft and not distended. Rather tender over gall-bladder with sense of resistance.

Operation.—March 13, 1909. Dr. Deaver. Ether. Upper right rectus incision, intestines walled off; small, thickened gall-bladder full of stones revealed. Gall-bladder opened and stones removed. No bile present. Several very hard nodules in liver about fundus of gall-bladder. Piece of tissue removed from liver nodule for diagnosis. Surrounding common duct and involving major portion of gastro-hepatic omentum was a hard mass. Gall-bladder sutured with iodine gut and wound closed with iodine gut in layers. No drainage. Uneventful recovery surgically, but still has much pain at times.

Urine.—Negative. Culture from gall-bladder sterile.

Blood.—Hb., 82 per cent.; red, 3,720,000; W. B. C., 8750; polymorphonuclears, 69 per cent.; coagulation time, four minutes.

Stool.—Liquid containing large green and brownish fæcal masses. Bile present. Occult blood negative. Cammidge reaction negative.

Pathological Diagnosis.—Carcinoma.

CHAPTER V.

INJURIES OF THE LIVER AND BILIARY PASSAGES.

Injuries of the Liver.

The liver is more often injured than any of the solid abdominal viscera, though scarcely so often as the intestinal tract. It is predisposed to injury (1) by its *size*, especially if this is increased by disease; (2) by its *position* in contact with the anterior abdominal wall and ribs in front, with the vertebral column behind, and with the diaphragm above; (3) by its *consistency*, being naturally inelastic, and having its friability increased by disease; and (4) by its relative *immobility*. Notable is the fact that 83 per cent. of sixty-five patients with injury of the liver, reported by Boljarski, had been drinking or were actually intoxicated when they received their injuries.

In a series of 365 cases of subcutaneous injury of the solid abdominal viscera, studied by Edler, the liver was injured in 189 cases, while the pancreas, spleen and kidneys, all combined, were injured in only 176 instances. Of 116 penetrating wounds, the liver was involved in sixty-five, while the pancreas, spleen and kidneys combined were involved in fifty-one cases.

Injuries of the liver may be classed as *subcutaneous* (ruptures), and *percutaneous* (stab and gunshot wounds). The relative frequency of ruptures and of stab and gunshot wounds varies considerably with the geographical location of the patients. In this country rupture is the most frequent form of injury, and this appears also to be the case in Germany, as well probably as in Great Britain and France. But in St. Petersburg Boljarski found ruptures comparatively rare.

RELATIVE FREQUENCY OF LIVER INJURIES.

Author.	Rupture.	Gunshot Wounds.	Stab Wounds.	Total.
Boljarski (Russia).....	8	2	55	65
Finsterer (Germany).....	25	11	4	40
Tilton (New York City).....	12	9	4	25

Uncomplicated injuries of the liver are much less rare than are uncomplicated injuries of any other viscus in the upper abdomen. Complicating injuries oftenest involve the pleura, diaphragm or lung; less often the kidneys, stomach or pancreas. Ruptures are much more often uncomplicated than are gunshot or stab wounds. In Boljarski's series of sixty-five cases there were the following complicating injuries:

Abdominal wall	57 cases.
Diaphragm	18 cases.
Pleura	18 cases.
Ribs	12 cases.
Stomach	6 cases.
Bowels, Mesentery, Pancreas, Spleen, each	1 case.

Prognosis.—The mortality recorded in most statistics is too low, since it is deduced from collected cases, and not from a large and consecutive series. Among isolated case reports favorable results always unduly predominate. The following classical statistics may be perused in this connection:

CASES OF INJURY OF THE LIVER (NO OPERATION).

Mayer (1872)	276 cases	59 per cent. mortality.
Otis (1876)	181 cases	62 per cent. mortality.
Edler (1887)	104 cases	57 per cent. mortality.

Edler collected in all 543 cases of liver injury, treated without operation: 189 ruptures, with 162 deaths; 289 gun-shot injuries, with 159 deaths; and 65 stab wounds, with 42 deaths; a total mortality of 66.8 per cent.

OPERATIONS FOR INJURY OF THE LIVER.

Terrier and Auvray (1896)	56 operations	32 per cent. mortality.
Terrier and Auvray (1901)	42 operations	23.6 per cent. mortality.
Giordano (1902)	257 operations	33.5 per cent. mortality.

The first operation for hemorrhage from the liver was done in 1887 by Burckhardt, in a case of stab wound; the wound was packed and the patient recovered.

The prognosis of the three main varieties of liver injury is indicated by the following statistics:

STATISTICS OF OPERATIVE TREATMENT OF INJURIES OF THE LIVER

Author.	Ruptures.				Gunshot Wounds.				Stab Wounds.			
	Total	Rec.	Died.	Mort. Per Cent.	Total.	Rec.	Died.	Mort. Per Cent.	Total.	Rec.	Died.	Mort. Per Cent.
Boljarski	8	0	8	100	2	1	1	50	55	38	17	40
Hagen	12	3	9	75	....	....	....	...	7	....	...	.....
Wilms	15[1]	3	12	80	....	....	....	...	....	....	...	.....
Finsterer	6[2]	4	2	33	1	1	0	0	2	2	0	0
Tilton	....	....	....	62.5	....	....	....	33	....	....	...	28.5
Thöle (all collected cases to 1911).	260	100	160	61.5	200	102	98	49	292	220	72	24.6

The great importance of *early operation* is indicated by the following statistics of Thöle:

MORTALITY AFTER OPERATIONS FOR INJURIES OF THE LIVER.

	Rupture. Mortality Per Cent.	Stab Wound. Mortality Per Cent.	Gunshot Wound Mortality Per Cent.
1. Operation within six hours	39.5	14.5	32.2
2. Operation from seven to twelve hours	50.4	20.7	45.5
3. Operation from thirteen to twenty-four hours	66.6	33.3	50.3
4. Operation after twenty-four hours	86.3	50.0	75.4

Rupture of the Liver.—The causes are *direct* and *indirect* violence. Blows, falls and crushes are the usual forms of direct injury; usually they produce stellate tears. Indirect injuries usually are effective only when the liver already is the seat of disease, though in falls upon the feet or buttocks the liver may be ruptured by "counter-stroke," or it may burst in the sagittal plane from being bent upon itself. Hubbard quotes Henzelman's statistics of 151 subcutaneous injuries of the liver, one-third of which were caused by indirect violence. Though direct violence

[1] Four other patients, not included in the above, died of hemorrhage before operation could be done.

[2] Two other patients, not included in the above, recovered without operation. Presumably they had a subcapsular or central rupture.

usually acts from the front, injury may be due to falls on the back, as in Houghton's patient; to lateral pressure (Herzog), or even to muscular action (Waring). In Herzog's patient the accident occurred as the result of an obstetrical practice said to be common in the Philippine Islands: the natives place a folded cloth around the loins of the woman in labor, and one or two persons make traction upon it. This patient died, and at autopsy a rupture of the liver was found, due to the eleventh rib being forced into the liver substance.

Ruptures are much more frequent in the right than the left lobe of the liver, and upon its upper surface than upon its lower. In 182 cases analyzed by Thöle the right lobe was affected six times as often as the left.

Ruptures of the liver are divided by systematic writers into three classes:

1. Rupture of the hepatic tissue involving the capsule.

2. Separation of the unruptured capsule from the liver substance, with subcapsular hæmatoma.

3. Central ruptures, giving rise to separate or confluent hæmatomata, which may develop into cysts or abscesses.

The first class is the most important, owing to the necessary occurrence of intraperitoneal hemorrhage, with its attendant dangers. The second and third classes may prove little more serious than a contusion, unless the capsule of Glisson eventually ruptures, or unless secondary infection of the hæmatoma occurs.

Ruptures involving the capsule of Glisson may be single or multiple; superficial or deep; linear, stellate, or gaping; the liver substance may be pulpefied; portions of liver may be detached or the entire organ may be torn in half (Fig. 18).

Symptoms.—Immediately after the injury there usually is shock, with nausea and vomiting, followed in most cases by symptoms of internal hemorrhage. The pulse may vary considerably, being very slow very soon after the injury but soon increasing in frequency. Finsterer observed marked bradycardia in three out of eight cases of injury of the liver by contusion, and he made experimental studies to prove the value of this symptom in diagnosing rupture of the liver. Bradycardia occurs in some cases of cholæmia, and Finsterer

claims it is the result of the action of bile-acids, and states that even if on account of excessive hemorrhage the pulse does not become slower yet the absorption and elimination of bile-salts may be detected by appropriate tests of the urine. He claims that the pulse is the most reliable guide in those cases where internal hemorrhage is not severe, and where rigidity of the abdominal muscles possibly may be thought to be due to a parietal injury without visceral complication. Particularly if the bradycardia develops while the patient is under observation,

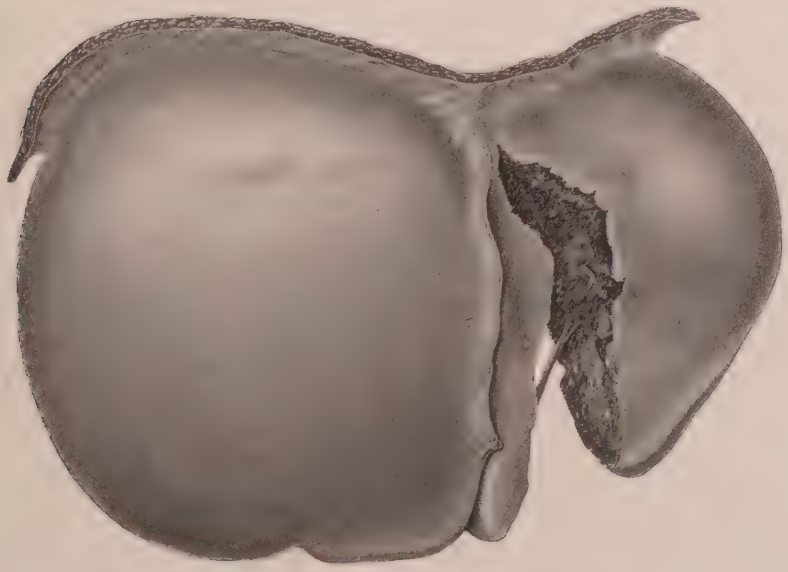

Fig. 18.—Rupture of the Liver; from a Patient in the Episcopal Hospital. (*See Case History, page 242.*)

or if it is present at first, only to vanish as hemorrhage progresses, should injury to the liver be suspected. In one of Finsterer's patients the pulse was forty-eight, and in another fifty-two at the time of the first examination. Thus a slow pulse is of considerable diagnostic importance, though a rapid pulse does not by any means exclude visceral injury. He has collected, in all, thirteen cases of marked bradycardia due to this cause. Thöle found bradycardia recorded in exceedingly few of his collected cases, and thinks no significance should be attached to it.

The abdomen at first is soft and flat, and in some cases may

remain so for hours; usually, however, it very soon becomes rigid and board-like, and later distended. Tenderness is more or less general soon after the injury but gradually becomes localized over the hepatic region. The respirations become shallow and thoracic in an endeavor to keep the liver at rest. As hemorrhage increases they become sighing in character. Generally there is abdominal pain, and very often pain in the back. The liver dullness may be greatly increased, and in some cases of profuse bleeding shifting dullness in the flanks may be detected. Jaundice may occur after a day or two, especially if operative treatment has not been undertaken promptly; but as a rule it is of no diagnostic importance.

The *diagnosis* of rupture of the liver often is difficult, especially soon after the injury has been received. A history of injury in the hepatic region always should make one suspect rupture of the liver. Hubbard lays stress on the effect of heat on the abdominal muscles as a differential diagnostic sign. In simple contusions of the abdominal muscles these relax after the application of heat, while they remain rigid and the degree and extent of the rigidity increases when there is an intra-abdominal traumatism. Localization of tenderness and of spasm of the abdominal muscles often points to the organ involved. Jaundice coming on two to four days after a suspected injury of the liver, the result of absorption of be through the perilitoneum, is seen in about 20 per cent. of the cases. Jaundice appearing much later generally is due to abscess formation in the liver.

Prognosis.—The prognosis in rupture of the liver is greatly modified by the treatment instituted and the length of time elapsing between the injury and operation. Edler found that more than half of 543 collected cases of rupture of the liver died from hemorrhage within twenty-four hours following the accident. The total mortality in this series of collected cases, none of them treated by operation, was 66.8 per cent. The diagnosis, in the case of patients who recover without operation, usually is rather uncertain, and Mercadé probably was within the truth when he claimed a death rate of 80 per cent. without operation. It cannot be denied that recovery may occur without operation, especially in the cases of subcapsular or central rupture; but even in these cases a deferred operation sometimes has had to be under-

taken for hepatic or subphrenic abscess or some other sequel of the original injury. According to Finsterer such cases have been recorded by Graser, Fertig and Chiari.

Much more reliable information is derived from statistics which comprise series of *consecutive cases* from one or several hospitals. Such series are presented in the following table:

SERIES OF CONSECUTIVE OPERATIONS FOR RUPTURE OF THE LIVER.

Bartels (1904)	6 operations	83 per cent. mortality.
Boljarski (1911)	8 operations	100 per cent. mortality.
Finsterer (1912)	42 operations	69 per cent. mortality.
Hagen (1906)	12 operations	75 per cent. mortality.
Wilms (1905)	15[1] operations	80 per cent. mortality.

In those cases not subjected to immediate operation the prognosis is modified, naturally, by the extent of the injury, the amount of hemorrhage, and the presence or absence of infection. Usually there is extravasation of bile into the peritoneal cavity. This causes little damage if it is sterile, but when it contains bacteria, or when it is allowed to remain indefinitely in the peritoneal cavity and becomes infected by migration of bacteria from the intestines, spreading peritonitis follows. The immediate danger following the injury is from hemorrhage; the remote danger is from sepsis.

A few well-authenticated cases of *embolism* by pieces of liver tissue have been recorded. Finsterer mentions the fatal cases reported by Marshall, Hess, Schmorl, Zenker, and Williams. Schnitzler thought the pulmonary symptoms in his patient, who recovered, were due to an embolus of hepatic tissue.

The *treatment* of diagnosticated or strongly suspected rupture of the liver consists in immediate laparotomy with repair of the lesion in the liver. We believe with Noetzel that shock is not a contraindication to immediate operation. Hubbard believes that it is better to treat the shock by means of heat, morphine, strychnine, and saline solution, before opening the abdomen. As the greatest danger comes from the hemorrhage, and as the liver tissue tends to prevent collapse of the bleeding vessels, we feel quite certain it is safer to subject the patient to the added burden of an operation than to the added burden of continuing hemorrhage.

In some cases, however, death occurs so rapidly that there is

[1] Four other patients died before operation could be performed.

no opportunity for operation. This was true in the patient whose liver is represented in Fig. 18.

Rupture of the Liver; Death.

A man aged thirty-two years was admitted to the Episcopal Hospital, service of Dr. R. H. Harte, Nov. 13, 1899. He had been caught between the back of a wagon and the pole of another wagon, the pole striking the patient in the upper abdomen. There was slight discoloration of the skin posteriorly at the level of the eighth thoracic vertebra. The patient was in profound shock when admitted, unconscious, and with symptoms of internal hemorrhage. Death occurred ten minutes after admission.

Autopsy.—Peritoneal cavity full of fluid blood; left lobe of liver nearly detached from right by rupture running parallel to the great transverse fissure and about three-quarters of an inch to its left. Only a narrow isthmus of liver tissue holds the left lobe to the right, this isthmus being at the posterior margin.

The abdominal incision should be made near the median line in the epigastric region, through the right rectus muscle. The convex surface of the liver may be reached by section of the suspensory ligament, or by resecting, the eighth, ninth, and tenth costal cartilages and drawing the ribs outward, as described in Vol. I. page 324. A large incision may be required, and if one from the ensiform to the umbilicus near the median line does not suffice, the best exposure will be secured by transverse division of the rectus at the level of the umbilicus, as in Czerny's gall-bladder incision (page 443).

Hemorrhage may be controlled by suture or by packing the wound in the liver with gauze. Temporary clamping of the pedicle of the liver to secure hæmostasis was proposed by Pringle, according to Finsterer; and Baron devised a special clamp for the purpose.[1] One blade of the forceps is passed through the foramen of Winslow and the other in front of the gastro-hepatic omentum. Such a measure is seductive in theory, but we should be fearful of injury to the portal vein. The blood-pressure in the liver is very low, and if the surgeon does not lose his head on encountering such profuse hemorrhage, very moderate pressure with gauze on the bleeding surface is sufficient to check it.

It is perfectly feasible to suture most wounds of the liver without having bleeding along the suture tract, *provided the wounds are*

[1] McDill's experiments are referred to at page 475.

accessible. In the case of lacerated wounds, where the hepatic tissue is pulpefied, it may be impossible to make sutures hold. Various methods of liver suture are described in Chapter IX.

Tamponade of the wound with gauze almost always is much easier than suture, but it is a method that entails a long convalescence and predisposes to infection. Boljarski found that for patients whose liver wounds had been tamponed the average stay in the hospital was sixty days. This was the case in the patient whose history is appended.

RUPTURE OF LIVER; TAMPONADE; RECOVERY.

H. G., male, aged twenty-eight years, admitted to the German Hospital November 9, 1909 At 3 P. M. on the day of admission had been caught between two heavy objects, crushing the lower part of the chest antero-posteriorly. *On admission,* half an hour after injury the pulse was 84, temperature 98, and respirations 28. There was very evident fracture of the tenth and eleventh ribs on the right side. Small area of dulness in both flanks Liver dulness not obliterated. Very slight rigidity of the abdominal muscles. Peristalsis present. There was some tenderness over the hepatic region. One hour later there were signs of internal hemorrhage, the pulse was much more rapid, the skin and mucous membranes were becoming blanched, respirations were slightly sighing in character, and the area of dulness in the flanks was increasing. Immediate operation was advised, but was deferred by patient until 7.30 P. M., four hours and a half after the injury.

Operation by Dr. Ross. Ether anæsthesia. Abdomen opened through upper right rectus. Peritoneal cavity filled with blood. Linear tear found on convex surface of right lobe of liver. The wound was about 3 inches long, very deep, and gaping. The wound in the liver was packed in layers, seven pieces of gauze being used. Blood was mopped out of peritoneal cavity, and the abdominal wound was closed, the gauze packing emerging at upper end. The patient was greatly shocked, and 800 c.c. of saline solution were given intravenously during operation; this amount was repeated three hours later.

After reacting the patient did well. The last piece of gauze was removed on November 15, sixth day after operation. The sinus healed slowly, but was entirely closed when the patient was discharged January 21, 1910.

At the time of admission the patient's hæmoglobin was 80 per cent. Two days later it was 56 per cent.

When a tampon is used it was formerly taught that it should not be removed in less than forty-eight hours, and that it should not remain in place longer than three or four days, for fear of inducing the formation of a persistent biliary fistula. But, as Bol-

jarski points out, secondary hemorrhage may occur up to the sixth or seventh day, or even later; and the vast experience in wounds of the liver acquired by this writer in Zeidler's clinique at St. Petersburg should have great weight in bringing surgeons to adopt his practice. In his series of cases of liver injury the tampon was used no less than sixty times, and he made it a practice not to loosen the pack for a week, and even then to draw it out only by degrees.

The actual cautery, and the use of live steam have been recommended for the control of hemorrhage from the liver, but such measures increase the dangers of secondary hemorrhage.

Omentoplasty as an aid in controlling hemorrhage from wounds of the liver was studied experimentally by Loewy, and has been used by Mauclaire in resection of the liver for tumor, and in a case of stab wound, with success. Boljarski used this method in five cases successfully: the omentum is stitched to the borders of the hepatic wound, or is stuffed into the wound, if large, and the wound margins are then sutured together over it. Boljarski used silk and ordinary intestinal needles. In three cases the abdominal wound could be closed without drainage, but in the two other patients drainage was necessary. The average stay in the hospital of these five patients was only twenty-four days, which compares very favorably with the average stay after tamponade of sixty days.

Gunshot Wounds of the Liver.—Owing to the position and size of the liver, gunshot wounds of that organ occur next in frequency to those of the intestine. In civil practice gunshot injuries of the liver represent from 23 to 33 per cent. of all gunshot wounds involving the abdomen; but in only about 12 per cent. of such wounds is the injury of the liver uncomplicated by lesions of other viscera. In war the liver is wounded in 50 per cent. of abdominal injuries, but only about 13 per cent. are uncomplicated wounds. The structures most often wounded in complicated cases are the pleura, lung, diaphragm, stomach and colon.

Gunshot Wound of Pericardium, Œsophagus, Diaphragm, Liver, and Lung. Combined Operation. Death from Pneumonia.

E. F., female, aged seventeen years, was admitted to Dr. G. G. Davis's service in the Episcopal Hospital, March 6, 1909. The bullet had entered the sixth left intercostal space close to the sternum, and lodged beneath the skin of the back in the eleventh left intercostal space, about 3 inches from

the vertebral line. Shock was present, and there were symptoms of internal hemorrhage and of wound of the lung. No evidence of injury to the heart. The abdominal muscles were so rigid as to indicate, in connection with the presumed course of the bullet, that there was perforation of the diaphragm with injury of the abdominal viscera.

Operation by Dr. Ashhurst, 2.15 A. M., five hours after injury. Ether anæsthesia. "Combined operation." (See Vol. I, p. 311.) Incision from tip of left eighth costal cartilage downward and inward to mid-line, obliquely across rectus muscle. Peritoneum opened, and a little blood found. Better exposure was secured by cutting across the eighth costal cartilage and splitting the diaphragm upward for 2 or 3 inches. In doing this the pleura was wounded and found full of blood. The pleural opening was temporarily closed by clamp. Good exposure was now secured of the under surface of the diaphragm, the fundus and cardia of the stomach, the left lobe of the liver, and of the spleen. There was no active hemorrhage, and no wound of the stomach, colon, liver or spleen was found. The diaphragm was sutured, and the abdominal incision closed around a gauze wick. The original skin incision was then continued outward in the seventh intercostal space to the posterior axillary line, and the pleura was widely opened. There was very great dyspnœa until the pleura was widely opened, whereupon the respirations became more tranquil. About 500 c.c. of fluid blood were evacuated from the pleural cavity, active hemorrhage continuing. On the pleural surface of the left dome of the diaphragm was a hæmatoma, about 7.5 cm. long by 2.5 cm. wide, presumably a grooved wound made by the bullet. The lower and posterior margin of the lung was full of blood, but when drawn into the wound with volsellum forceps no active hemorrhage was detected, and no sutures were inserted. There was bleeding from the wound of exit in the posterior parietal pleura. This was tamponed, and the thoracic wound was closed around the gauze wick. The bullet was then removed from its subcutaneous position by another incision.

Death in fifty-three hours from double septic pneumonia.

Autopsy.—The bullet entered the sixth left intercostal space and penetrated the pericardium without wounding the heart; there was very little blood in the pericardium. On leaving the pericardium the bullet entered the diaphragm, *grooving* the œsophagus as the latter passed through the diaphragm. The bullet then perforated the thin margin of the left lobe of the liver, making a tunnel 1 inch long. It then re-entered the diaphragm, penetrated the pleura, passed through the lower border of the lung, and left the pleura in the eleventh intercostal space. There was no blood in the peritoneal cavity and no peritonitis; no pus and little blood in the pleural cavity. Death from diffuse pneumonia involving both lungs.

The immediate effect of perforation of the liver by a bullet is hemorrhage. The extent of the hemorrhage will be modified by the size of the wound, the location and the course of the bullet, and somewhat by the period of digestion, since the hepatic

blood-vessels become enormously engorged during the process of digestion. Gunshot wounds when the liver is in this condition are followed by more profuse hemorrhage than after digestion has been completed.

The *symptoms* of uncomplicated gunshot wounds of the liver are chiefly those of internal hemorrhage. Other symptoms are comparatively unimportant. Shock is not always marked. Pain may be severe or absent; it is an important symptom when referred to the right shoulder. As a rule muscular rigidity is not marked. Jaundice does not occur for some days; Edler says it develops in about 20 per cent. of the cases.

The *diagnosis* depends on the position of the wound of entrance of the bullet, and the direction in which it travelled, if this is known or if it can be ascertained from the wound of exit or the site of lodgment. Usually symptoms of hemorrhage are sufficiently marked to warrant a diagnosis of wound of the liver when the course of the bullet indicates this as a probability. In most cases the complicating injuries to other viscera are of greater importance from a diagnostic and surgical standpoint than the liver injury.

Prognosis.—Uncomplicated gunshot wounds of the liver are very rare. Finsterer could find reports of only twenty-six operations in such cases, and from this series he did not exclude wounds of the pleura or slight injuries of the lung which often are negligible. Nineteen of these twenty-six patients recovered, including one operated upon by Finsterer himself; and seven died, a mortality of 27 per cent. Where the liver injury is complicated by gunshot wounds of other viscera, the death rate is even higher. Among eleven consecutive cases from various German cliniques, reported by Finsterer, there were three deaths (27.2 per cent.); of Boljarski's two patients, one died (50 per cent.).

The *treatment* in all cases of gunshot wound of the liver in civil life is immediate operation. Expectant treatment might be justified in those few cases where the bullet is of small calibre, where there are no signs or symptoms of internal hemorrhage or of complicating injuries; or where no facilities exist for operation. But in all other cases operation should be undertaken before time has elapsed for signs of peritonitis to arise. Usually the signs of internal hemorrhage are so urgent that immediate

operation is required on this account. The wound of entrance should be disinfected, as advised in Vol. I, page 322, and the liver exposed as recommended in cases of rupture of this organ (Vol. II, page 242). The bleeding from the wound in the liver should be controlled by suture, packing, or omentoplasty.

Stab Wounds of the Liver.—The relative frequency of these injuries has been indicated at page 235. Among Boljarski's fifty-five cases there were thirty-two uncomplicated by injuries to other viscera.

Stab wounds are followed by profuse hemorrhage, the bleeding being much more marked than in gunshot wounds. The *symptoms* are those of hemorrhage, possibly with symptoms of complicating injury to other viscera, as in the case of rupture and gunshot wounds.

The *diagnosis* of stab wounds of the liver is based on the history, together with the location of the external wound in the region of the liver, and on the presence of hemorrhage. It is impossible to determine positively from the symptoms alone whether or not the liver has been injured. Under ordinary circumstances the surgeon must content himself with the diagnosis of internal hemorrhage.

The *prognosis* after prompt operation is comparatively favorable. A few patients may die before opportunity for operation is afforded. Operative treatment was adopted in all of Boljarski's fifty-five patients: seventeen deaths occurred, a mortality of 40 per cent. Among the thirty-two uncomplicated cases, however, there were only four deaths (12.5 per cent.); while of the twenty-three cases complicated by other abdominal or serious thoracic injuries, no less than thirteen terminated fatally (56 per cent.).

The *treatment* consists in immediate operation. If the fact of penetration of the abdominal cavity is uncertain, the surgeon should explore the wound with the precautions advised in Vol. I, page 316, for the reasons there stated. In cases where the fact of penetration is assured by the prolapse of abdominal contents (a condition met with in fifteen out of Boljarski's fifty-five cases), the peritoneal cavity may be opened at once. If the stab wound in the abdominal parietes is in a convenient location, the operative incision may be made through it. It is better when possible to operate through the stab wound than to make a new incision,

although the latter of course is to be preferred if it facilitates rapid work and allows more convenient treatment of the rent in the liver. The latter should be closed by suture, packing or omentoplasty, as described at page 244.

THREE STAB WOUNDS (SUICIDAL) OF THE LIVER. SUTURE. RECOVERY.

G. S., male, Russian, aged thirty years, a tailor by occupation, attempted to kill himself at 1.30 P. M., January 5, 1912, by stabbing himself with his long tailor's shears. He was brought to the Episcopal Hospital, and admitted to Dr. Frazier's service. Examination at 5 P. M. showed that there was no shock. The pulse was 120 to 130, considerably faster than on admission, when it was about 100. He was rather pale, and quite impassive. There was no tenderness or rigidity of the adbomen. There were five stab wounds in the epigastric region. The largest, about 4 cm. long, was just to the right of the mid-line, and close to the costal margin, dividing the fibres of the right rectus muscle transversely. No prolapse of abdominal contents. No free fluid in the flanks.

Operation by Dr. Ashhurst, at 5.30 P. M., four hours after injury. Ether anæsthesia, preceded by nitrous oxide. Gloved finger inserted into largest stab wound entered peritoneal cavity and recognized the liver immediately beneath the wound. Incision through right rectus muscle, close to median line, passing through the largest of the stab wounds. Free blood was present in the peritoneal cavity. The stomach and omentum were packed off with gauze, and the incision extended up beside the ensiform cartilage. On the upper convex surface of the right lobe of the liver, about 3 inches from its anterior border, there was a stab wound, bleeding actively. By elevating the patient's lumbar spine, this wound became accessible, and was closed with one mattress suture of chromic catgut, in a curved intestinal needle. Many clots and some fluid blood sponged away from right subphrenic space. Through a stab wound in the suspensory ligament of the liver a flood of blood came from the left lobe. The suspensory ligament therefore was divided as far back as the lateral ligaments of the liver, and large quantities of fluid and clotted blood were evacuated and wiped away from above the left lobe. By depressing the liver two more stab wounds were found, both on the upper surface of the left lobe, neither penetrating to its under surface. Only one was bleeding actively. This was closed with one mattress suture of chromic catgut. The left subphrenic space was wiped dry. The stomach, gall-bladder, pylorus, omentum, and transverse colon were examined, but no further lesions were found. The suspensory ligament of the liver was then repaired by sutures, thus reattaching the liver to the diaphragm. The abdominal wound was closed without drainage. The smaller stab wounds were tamponed. The operation lasted fifty minutes, and during its performance 1000 c.c. of saline solution were administered intravenously.

Culture from the blood evacuated during operation (about 500 c.c.) remained sterile.

Recovery was slow owing to pulmonary complications. For signs of effusion in the pleural cavities (Dr. Geo. W. Norris) aspiration was done on both sides on January 22, and on the right side on January 28 and February 7. But at no time was fluid found.

The patient developed melancholia. He could not be made to eat. His abdominal wounds were slow in healing. He was finally discharged, sound physically and mentally, on May 11, 1912, more than four months after his attempt at suicide. For the last month of his stay in the hospital he had done much work in the ward, helping the orderlies, etc.

Injuries of the Gall-bladder and Biliary Passages.

Injuries to the gall-bladder and ducts occur less frequently than injuries to the liver.

Subcutaneous ruptures are the commonest. They are very seldom coincident with injuries to the liver; among the 260 cases of ruptue of the liver collected by Thöle, there were only two instances where the gall-bladder also was ruptured. Thöle adds thirteen cases of rupture of the gall-bladder or bile-ducts to the sixty-three cases collected in 1903 by Lewerenz. Previous disease of the biliary tract predisposes it to rupture. A distended gall-bladder such as is often seen in the presence of an obstructed cystic duct may readily be ruptured by a slight fall on the side, or by a contusion.

The fundus of the gall-bladder, which is the most exposed portion of the bile-passages, is most frequently injured. Among the sixty-three cases of rupture of the biliary passages collected by Lewerenz, the site of the lesion was

In the gall-bladder	24 cases
In the choledochus	10 cases
In intra-hepatic bile ducts	8 cases
In the hepatic duct	6 cases
In the cystic-duct	1 cases
Not mentioned	14 cases

The injuring force may also cause complicating injuries of the adjacent structures, such as the liver, stomach, intestine, etc. Kilgour reported a case in which a portion of the liver with its attached gall-bladder was completely separated from the rest of the liver. Paget reports a case in which the patient was run over by a sulky. The liver had been torn from its suspensory ligament and rotated forward. The anterior edge of the liver was flattened

out and adherent to the transverse colon. The gall-bladder had been torn from its bed in the liver and was suspended by a pedicle formed by the cystic duct and vessels. Bile was being discharged from a tear in the common duct. The patient recovered after two operations, death being prevented, according to Paget, by hypodermic injections of pituitary gland.

The early *symptoms* presented by rupture of the biliary passages are similiar to those of other internal injuries: pain, shock, vomiting and restlessness. Signs of hemorrhage are not so marked as after rupture of the liver, as bleeding never is so profuse. Peristalsis may be present or absent. Dullness may be detected in the right flank and later in the left flank. Vomiting in some cases has been continuous. The abdomen becomes distended and if the bile is so infectious as to cause peritonitis, the abdomen becomes tympanitic. Sterile bile, while it may give rise to very decided symptoms, does not cause fatal peritonitis. If the bile is allowed to remain in the peritoneal cavity for a considerable length of time, the intestine becomes covered with a fibrinous membrane which may be peeled off (Kehr). Kehr states that as much as twenty quarts of bile have been known to collect in the abdominal cavity. As the bile is being absorbed by the peritoneum, generally after the third or fourth day, symptoms of cholæmia develop.

The picture presented later will be modified by the presence or absence of infection. The bile is a good culture medium and the presence of micro-organisms will give rise to a wide-spread peritonitis. Bacteria may enter the peritoneal cavity from the duodenum through the rent in the bile-passages; or if there was cholangeitis immediately prior to the injury almost invariably peritonitis will ensue.

The *diagnosis* is based on the history of injury to the abdomen or lower thorax, with a train of symptoms which points to visceral injuries. As pointed out by Robson the most characteristic sign of injury to the bile-passages is the jaundice which usually appears in three or four days, due to the reabsorption of the extravasated bile. This jaundice is not noted so frequently in rupture of the liver. In a few instances the only thing suggesting injury to the biliary tract is the history of a blow or injury in the right hypochondrium; the other signs and symptoms are those following any

internal injury with the exception of signs of excessive hemorrhage. The following case illustrates some of the conditions found at time of operation:

Rupture of the Gall-bladder. Cholecystostomy. Recovery.

J. T., male, aged thirteen, admitted to the Mary J. Drexel Home, December 29, 1906. Three days before admission he had been kicked in the upper abdomen while playing basket ball. He was unconscious for a few moments, and began vomiting one hour after receiving the injury and continued vomiting everything ingested for the next forty-eight hours. On admission patient had a temperature of 100° F., and a rapid pulse of good volume. Vomitus was greenish in color, with no odor. Urine was normal. There was no discoloration of the skin at the site of injury. Jaundice was not present. The abdomen was very much distended, the onset of this symptom having been noted thirty-six hours after the injury. There was no rigidity of the abdominal muscles. The boy complained of pain in the right hypochondrium, but there was no mass palpable and no area of tenderness could be detected. Both flanks were dull on percussion.

Operation by Dr. Deaver, December 29, 1906. On opening the peritoneum, there was a gush of a large quantity of greenish-yellow fluid, resembling bile. The entire abdomen and pelvis was filled with bile which was being discharged from a rupture in the fundus of the gall-bladder. The abdomen was flushed out with saline solution. There was no evidence of peritonitis. Cholecystostomy was performed and the pelvis was drained through a separate incision. No injury to any other viscus could be found. The patient made a good recovery.

The *prognosis* in rupture of the gall-bladder and ducts should be favorable in all cases where early operation is performed. Of twenty-nine cases in which no operation was done, collected by Edler, twenty-two died. Septic peritonitis will follow in all cases where the bile in the peritoneal cavity becomes infected, and the prognosis under such conditions is much more grave. Even where the bile as originally effused is aseptic, it serves as such a good culture medium that its secondary infection from the intestinal tract or through the blood stream is very probable. In Courvoisier's series of thirty-four cases of subcutaneous rupture of the biliary passages, death occured in twenty-two, in five from collapse within the first thirty hours and in seventeen from peritonitis and the toxic action of the absorbed bile. He also reported in his fourteen cases of injury due to penetrating wounds three deaths from collapse and six from sepsis. Uncomplicated injury of the biliary

passages allows a much more favorable prognosis than the complicated cases. In the latter instances the injuries to other viscera are of far more moment than the injury to the gall-bladder or ducts.

The *treatment* consists in immediate laparotomy with repair or drainage of the injured part. If the diagnosis is uncertain we believe it is much safer to explore the abdomen through a small incision than to wait until a positive diagnosis of internal injury is made from the presence of jaundice, a marked distention of the abdomen, the presence of spreading peritonitis, etc. Incision of the abdominal walls with inspection of the viscera we believe to be better surgery than removal of the fluid contents of the peritoneal cavity by means of the aspirating trocar and canula. Courvoisier reckoned that repeated aspiration might cure 33.3 per cent. of patients, while 66.6 per cent. would perish from peritonitis or cachexia. Terrier and Auvray collected seventeen cases of rupture of the biliary passages in which aspiration, at times repeated, had been performed, ten of the patients recovering. Unless the rent in the bile-passages is very small so that it may be closed by plastic exudate in a short time, aspiration will not remedy matters as more bile will be constantly poured into the abdomen and absorbed. Aspiration can only remove the bile from the peritoneal cavity; it does not effect a closure of the rent in the injured structures.

When the opening in the gall-bladder is small, it may be closed and the external wound closed without drainage after evacuation of most of the fluid found in the abdomen. Flushing or irrigation of the peritoneal cavity is to be condemned. When the rupture is of considerable extent, the gall-bladder should be drained; or if the gall-bladder has been badly torn or shows the presence of marked disease, it should be removed. When the common duct is ruptured the opening, if small, may be closed; if the rent is large, drainage by means of a rubber tube should be instituted. If the entire lumen has been severed, the posterior wall of the duct may be sutured, with drainage by means of a T-shaped tube from the remaining opening in the anterior portion; or some form of anastomosis between the bile-tract and intestine may be done. In all cases, however, in which there is evidence of cholecystitis or cholangeitis, drainage should be instituted, and all at-

tempts at radical operation postponed until the patient becomes convalescent.

Penetrating wounds of the gall-blader are quite rare. Thöle gives references to seventeen cases in which operation was done: thirteen patients recovered, and four died, a mortality of 23.5 per cent. He found no published cases of open wounds of the bile-ducts.

REFERENCES.

Baron: Zentr. f. Chir., 1910, xxxviii, 1547.
Boljarski: Arch. f. klin. Chir., 1911, xciii, 507.
Burckhardt: Zentr. f. Chir., 1887, xiv, 88.
Edler: Arch. f. Klin. Chir., 1887, xxxiv, 173, 343.
Eisendrath: Jour. Am. Med. Assoc., 1902, ii, 1091.
Finsterer: Deutsch Zeit. f. Chir., 1912, cxviii, 1.
Fraenkel: Beitr. z. Klin. Chir., 1901, xxx, 418.
Hagen: Beitr. z. klin. Chir., 1906, li, 529.
Henzelman: Inaug. Dissert., München 1886, quoted by Hubbard, loc. cit.
Herzog: Surg., Gyn. and Obst., 1907, iv, 741.
Houghton: Military Surgeon, Carlisle, Pa., 1907, xxi, 63.
Hubbard: Bost. Med. and Surg. J., 1906, clv, 671.
Kehr: System of Practical Surgery (v. Bergmann, Bull. et al.), Phila. and N. Y., 1904, vol. iv, p. 632.
Kilgour: Edinb. Med. and Surg. Jour., 1841, lv, 352.
Loewy: Thèse de Paris, 1901.
Loewy: Rev. de Gyn. et de Chir. Abd., 1906, x, 821.
Mayer: Die Wunden der Leber und Gallenblase, München 1872.
Mercadé: Rev. de Chir., 1902, xxv, 89.
Nötzel: Beitr. z. klin. Chir., 1906, xlviii, 337, 1909, lxi, 215.
Paget: Austral. Med. Gaz., 1912, xxxi, 54.
Robson: Diseases of Gall-bladder and Bile-ducts, London, 1904, 3d Ed., p. 49.
Terrier and Auvray: Rev. de Chir., 1896, xvi, 717.
Thöle: Verh. d. Deutsch. Ges. f. Chir., 1905, xxxiv, i, 228.
Thöle: Deutsch. Zeit. f. Chir., 1909, ci, 533.
Tilton: Annals of Surgery, 1905, i, 20.
Waring: quoted by Douglas: Surg. Dis. of Abdomen, Phila., 1903, p. 643.
Wilms: Deutsch. med. Woch., 1901, xxvii, 569; 593.

CHAPTER VI.

SURGERY OF THE PANCREAS.

Historical.—The term pancreas, from the Greek πᾶν κρέας, meaning literally "all flesh," is found in the writings of Galen; and it was thought by Haller that there was a reference to it in the apocryphal work of Hippocrates (περὶ ἀδένων). According to Galen, an anatomical controversy arose over the pancreas between Herophilus and Eudemus (fourth century B. C.), the latter of whom stated that a fluid emptied out of the pancreas into the bowels and aided in digestion; yet this interesting fact was entirely forgotten under the influence of the humoral pathology of Galen (second century A. D.), who followed the teaching of Aristotle that the pancreas acted as a cushion or buffer for the stomach and the neighboring vessels. Even the early anatomists, such as Vesalius, Fallopius, and Bauhinus knew nothing of the function of the pancreas, though it is said that Alberti and Heurnius made it the subject of dissertations. Wirsung (1642) described the duct which bears his name, but his contemporary, Asellius, confused the pancreas with the retroperitoneal or mesenteric lymphatics; while some held that it acted as a filter for the secretions of the liver and the spleen.

In 1663 Regnei de Graaf published a work on the nature and use of pancreatic juice; he was also the first to describe pancreatic calculi. This was long before Santorini's work, which, however, was not published until 1775, thirty-eight years after the author's death. During this period (seventeenth and eighteenth centuries) the pancreas was associated in the medical mind with such diseases as melancholy, hysteria, and epilepsy. Such ideas were soon abolished, however, by the physiological and pathological discoveries of the nineteenth century. Claude Bernard described the pancreatic ferment in 1856, and the pathological anatomists of the day, Cruveilhier, Rokitansky, Virchow, v. Recklinghausen, and later, Orth, Langerhans, and Dieckhoff, laid the foundation of our present knowledge of pancreatic diseases by studying the gland *post-mortem*.

Hemorrhage in connection with diseases of the pancreas, the so-called "pancreatic apoplexy," was first noted in 1866 by Spiess.

Fat necrosis accompanying acute pancreatitis was first described by Balser, who noticed it first at autopsy in 1879; but the publications of Fitz, beginning in 1889, were the first to make the profession realize the importance of the acute affections of the pancreas.

Though the connection between pancreatic disease and diabetes was suggested by many writers[1] (Chopart, 1791; v. Recklinghausen, 1864; Lancereaux 1877), it was not firmly established until the publication in 1889 of v. Mering's and Minkowski's experimental work.

Gussenbauer, in 1882, was the first to operate successfully by laparotomy in a case of pancreatic cyst,[2] though Lücke (1867) and Rokitansky (1881) had previously done such operations unsuccessfully, and Thiersch (1881) and Kulenkampff (1881) had opened pancreatic cysts with success, in two stages. But the true Father of Pancreatic Surgery is Nicholas Senn, who, in 1886 published his experimental and clinical researches, with the object, as he modestly stated, of laying a foundation for the rational treatment by surgical means of some of the diseases of the pancreas. In 1896 Riedel first recognized chronic interstitial pancreatitis at operation; but it is chiefly to the teaching and example of Mayo Robson, from the year 1900 on, that modern pancreatic surgery owes its inspiration and guidance.

REFERENCES.

Alberti: De morbis mesenterii et pancreatis, Dissert., Wittenberg, 1578.[3]
Balser: Arch. f. path. Anat., 1882, xc, 520.
Bernard, Claude: Suppl. aux. Comtes Rend. des Séances de l'Acad. des Sc., Paris, 1856, i, 379.
Cawley: London Med. Jour., 1788, ix, 286.
Chopart: Malad. des Voies Urinaires, Paris, 1791, Tom. i, p. 53.
Claessen: Krankh. d. Bauchspeicheldrüse, Köln, 1842.

[1] Cawley (1788) is quoted (by the name Cowley) by many recent writers on the pancreas as one of the earliest to recognize this condition. Though the pancreas of his patient was full of stones, he distinctly states his belief that the seat of diabetes is in the kidneys, and the lesions of the liver and pancreas are the result, not the cause of that disease.

[2] Pancreatic cyst was first studied by Engel in 1841.

[3] This work has not been accessible to us.

Fitz: Bost. Med. and Surg. Jour., 1889, cxx, 181, 205, 229.
Galen: περὶ χρείας των μορίων, Lib. iv. Ch. 2; Ed. Kühn, Lipsiæ, 1822, iii, 344.
Galen: περὶ σπέρματος Lib. ii, Ch. 6; Ed. Kühn, Lipsiæ, 1822, iv, 646.
deGraaf, Regner: "De Succo Pancreatico: or a Physical and Anatomical Treatise of the Nature and Offices of the Pancreatic Juice; . . . written by D. Reg de Graaf, Physician of Delph, and Translated by Christopher Pack, Med. Lond." London, 1676. (The original work, published in 1663, has not been accessible to us.)
Gussenbauer: Arch. f. klin. Chir., 1883, xxix, 355.
Haller: Elementa Physiologiæ Corporis Humani, 1757–66; Tom. vi, Lib. xxii, f. 427.
Heurnius: De morbis mesenterii et pancreatis. Lugd. Batav., 1599.[1]
Hippocrates: περὶ ἀδένων. Ed. Kühn, Lipsiæ, 1825, i, 494.
Kulenkampff: quoted by Gussenbauer, loc. cit.
Lancereaux: Bull. de. l'Acad. de Méd., Paris, 1877, 2e Série, vi, 1215.
Lazarus: Zeit. f. klin. Med., 1903, li, 95.
Lücke: quoted by Gussenbauer, loc. cit.
v. Mering u. Minkowski: Centralbl. f. klin. Med., 1889, x, 393; Arch. f. exp. Path. u. Therap., 1889, xxvi, 371.
v. Recklinghausen: Arch. f. path. Anat., 1864, xxx, 363.
Riedel: Berl. klin. Woch., 1896, xxxiii, 1.
Robson, Mayo: Congr. Internat. de Méd., Paris, 1900; Sect. de Chir. Gen., p. 140.
Rokitansky: quoted by Gussenbauer, loc. cit.
Santorini: "Septemdecim Tabulæ quas nunc primum edit atque explicat . . . Michael Girardi." Parmæ 1775, Tab. xii, f. 143, et Tab. xiii.
Schirmer: Inaug. Dissert., Basel, 1893.
Senn: Trans. Amer. Surg. Assoc., 1886, iv, 99.
Spiess: Schmidt's Jahrb., 1867, cxxxiv, 270.
Thiersch: quoted by Gussenbauer, loc. cit.

Congenital Anomalies, Displacements, etc.—The pancreas very occasionally may be entirely *absent;* three such cases, associated with other congenital anomalies incompatible with life, are mentioned by Lancereaux. On the other hand, in a certain proportion of bodies there are so-called *accessory pancreases.* Letulle found this condition six times among two hundred bodies examined postmortem, while Opie found it only ten times in eighteen hundred autopsies. It is probable that systematic search might reveal one or more accessory pancreases in a fairly large proportion of autopsies. These accessory glands are usually of small size, comparable to a pea or bean, or even smaller, being very seldom so large as a nut; they are situated in the walls of the various

[1] This work has not been accessible to us.

portions of the alimentary tract, usually in the muscular coat; and they nearly always have clearly recognizable ducts. In a few instances islands of Langerhans have been present. In the cases collected by Robson and Cammidge, the accessory pancreas was situated in the stomach in eleven instances, in the duodenum in six, in the jejunum in eleven (nearly all close to its duodenal end), in the ileum in five (and always in association with an intestinal diverticulum, not Meckel's), and in a congenital umbilical fistula in one case. The accessory pancreas may occur as a single, isolated, glandular mass, or several masses may be distributed along the gastrointestinal tract. Several cases are on record of an accessory pancreas at the duodenal opening of the duct of Wirsung; and, in almost all individuals, lobules of pancreatic tissue surround the duct of Santorini as it passes through the duodenal wall (Opie, loc. cit., page 61). Usually all accessory glands are found at autopsy to be more or less diseased, and as the total amount of glandular tissue is very small it has been questioned whether it can in any efficient way supplement the functions of the pancreas itself.

That portion of the head of the pancreas which lies furthest to the patient's left sometimes becomes more or less separated from the rest of the gland by the superior mesenteric vessels, which emerge just above it from between the body of the pancreas and the transverse duodenum. This partially isolated portion of the head is sometimes described as the *Pancreas Minus*, but is not truly an accessory gland. Other portions of the pancreas occasionally are more or less isolated from the remainder of the gland, this condition being described by Glinski, who has studied the subject carefully, as *Pancreas Divisum*. That portion of the pancreas associated with the duct of Santorini, which arises as a separate outgrowth from the posterior wall of the duodenum, is that which is most often separated from the rest of the gland, which is developed as a twin outgrowth from the anterior duodenal wall. Normally it coalesces with the portion developing around the duct of Wirsung; and in the adult the body and tail of the pancreas are more apt to drain through the duct of Wirsung than through the duct of Santorini around which they were primarily developed.

The head of the pancreas may surround the duodenum completely, constituting the so-called *Ring-formed Pancreas* (*Pancreas Annulare*); Robson and Cammidge refer to nine such cases. In a few

instances this rare condition has simulated, soon after birth, infantile stenosis of the pylorus; Vidal successfully resorted to gastroenterostomy in such a patient three days after birth. Symptoms may not develop until adult life, when chronic pancreatitis, carcinoma, etc., affecting such an abnormally situated pancreas, may produce occlusion of the duodenum, which can be distinguished from pyloric stenosis only with the greatest difficulty. Lerat treated such a case, in a woman aged forty years, by partial pancreatectomy.

Displacement of the pancreas is very unusual, owing to its fixed retroperitoneal position. Sappey, according to Körte, claimed that tight lacing might eventually lead to displacement of the pancreas, by compressing the lower thorax. The tail is the least well fixed portion, and occasionally is dragged from its moorings by a wandering spleen (Helm and Klob; Estes; Runge). Solid tumors of the pancreas may also cause its displacement; in one-fourth of the seventeen cases studied by Finney, the tumor was freely movable.

Among the 276 cases of diaphragmatic hernia collected by Lacher, the pancreas was among the organs displaced into the thorax in no less than twenty-seven instances; while this was the case in two out of the twenty-six cases of congenital diaphragmatic hernia collected by O. Mayor in 1891, since the appearance of Lacher's paper. Such cases have very little surgical interest, so far as the pancreas is concerned, as any symptoms which may arise from interference with its function are sure to be overshadowed by the changes induced in the stomach and intestines.

The pancreas has been found three times in a *congenital umbilical hernia*, according to Körte; who also states that E. Rose and Rahn have each found it in a similar hernia in adults. Finally, reference must be made to the remarkable case observed in 1805 by Baud in which the pancreas formed part of a complicated case of *intussusception*. Guibert in 1829 described a somewhat similar case.

REFERENCES.

Baud: Jour. Gén. de Méd., Chir., et Pharm., 1805, xxiv, 20.
Estes: Medical News, 1882, xli, 119.
Finney: Annals of Surgery, 1910, i, 818.
Glinski: Arch. f. path. Anat., 1901, clxiv, 132.

Guibert: Jour. Univ. des Sc. Méd., 1829, liii, 155.
Helm u. Klob: Woch. d. K. K. Gesellsch. d. Aerzte zu Wien, 1856, ii, 597; cited by Körte, loc. cit.
Körte: Die Chir. Krankh. u. die Verletz. des Pankreas. Stuttgart, 1898, S. 225, 226 (Deutsche Chir., Lieferung 45d).
Lacher: Deutsch. Arch. f. klin. Med., 1880, xxvii, 268.
Lancereaux: Traité des Mal du Foie et du Pancréas, Paris, 1899, p. 800.
Lerat: Presse Med. Belge, 1910, lxi, 409.
Letulle: Comptes Rend., Soc. de Biol., Paris. 1900, lii, 233.
Mayer: Inaug. Dissert., Berlin, 1891; quoted by Körte, loc. cit.
Opie: Disease of the Pancreas, Phila. and London, 1903, p. 49, 61.
Robson and Cammidge: The Pancreas, its Surgery and Pathology, Phila. and Lond., 1907, p. 56.
Runge: Berl. klin. Woch., 1895, xxxii, 346.
Vidal: xviii Congr. Franç. de Chir., Paris, 1905, p. 739, Obs. iii.

Atrophy of the pancreas may exist, perhaps as a congenital deformity (hypoplasia), or as the result of fibrosis from hereditary syphilis. Byron Bramwell, Thomson, and Rentoul have described cases of arrested development, termed by Bramwell "pancreatic infantilism," characterized by persistent fatty diarrhœa, tympany, etc., which were much improved by the administration of pancreatic extract. Other forms of atrophy, as well as hyaline, fatty, and similar degenerations, usually are the results of chronic inflammation.

REFERENCES.

Bramwell: Scottish Med. and Surg. Jour., 1904, xiv, 321.
Rentoul: Brit. Med. Jour.,1904, ii, 1011, 1694.
Thomson: cited by Robson and Cammidge, "The Pancreas, its Surgery and Pathology," Phila., 1907, p. 132.

Infections of the Pancreas.

Pathogenesis.—*Infection of the pancreas* may occur in any one of four ways: (1) through the blood stream; (2) along the excretory ducts of the pancreas; (3) through the lymph-channels; and (4) by contiguity from neighboring structures.

1. *Infection through the blood* is considered rare. The pancreas is not situated as is the liver in relation to the portal circulation, and thus is not constantly inundated with hordes of bacteria from the intestines as we are now led to believe is the case with the liver (see page 5). The pancreas is thus almost immune

from infection through its venous channels, except by *retrograde embolism in portal thrombosis;* a case, possibly of this nature, was reported in 1885 by Musser, though it is not impossible that there was here an ascending sialodochitis. The pancreas occasionally is the seat of *pyæmic abscess*, as in the case reported by Roddick; or is diseased in other general infections; indeed the experimental and clinical facts recently adduced in support of hæmatogenous infection, by Abrami, Richet and Saint-Girons make it probable that this source is less rare than has been supposed hitherto. Macaigne observed a case of pneumococcic peritonitis with an abscess of the pancreas from which a pure culture of the *pneumococcus* was obtained; and though he expresses the conviction that the infection of the pancreas was received by way of its ducts, it seems quite as likely to have been hæmatogenic. S. Phillips observed a case of *scarlet fever* with pancreatitis and parotitis; and the occurrence of lesions in the pancreas as well as in the suprarenal glands, during the course of scarlet fever has recently been carefully studied by Tixier and Troisier. The association of *epidemic parotitis* (mumps) with pancreatitis is now well recognized, and though Fitz wrote over twenty years ago that "it may be safely stated that there is no reason for admitting the existence of a metastatic pancreatitis secondary to inflammation of the parotid gland," the belief is now very general, and we believe it is well established clinically, that such an affection does occur; but the pathogenesis of this metastatic pancreatitis is no more easily explained than the similar involvement of the testicles or ovaries. It was Schmackpeffer who, in 1817, first called attention to the fact that the pancreas might be involved in mumps. Gordon Sharp, who has recently studied the subject, points out that pancreatitis may precede as well as follow the parotitis. Ordinarily the symptoms of acute pancreatitis occur on the third or fourth day after the parotid swelling has reached its height; and the parotid may undergo resolution so soon as the abdominal symptoms appear. Sharp likewise calls attention to the fact that during an epidemic one patient in a household or in an infected area may show parotitis, while another patient may have only pancreatitis; but he contends that both are due to one and the same cause. If parotitis occurs in the course of pancreatitis, it seems much more reasonable to suppose, unless there

is an epidemic of the disease, that the infection reaches the parotid along its duct, from the mouth, as in typhoid fever, and as occasionally observed after etherization for various operations.

From the observations of Simonin, pancreatitis appears to be a rare complication of mumps; in 652 cases of the latter disease he carefully examined the abdomen for signs of pancreatic involvement, but found it only in ten cases (1.53 per cent.) Other observers, however, have found it much more prevalent in certain epidemics, as may be seen in the following table, which gives all the cases we have been able to find hitherto reported of pancreatic symptoms occurring in patients with mumps. So far as is known, the case of Lemoine is the only one which terminated fatally. In Edgecombe's patient the urine, examined by Cammidge himself, gave a positive "pancreatic reaction."

Author.	Cases of Pancreatitis Complicating Mumps.	Total Cases of Mumps Observed.
Auché (Jour. de Méd. de Bordeaux, 1905, xxxv, 769)	2	not stated
Cuche (Bull. Soc. Méd. des Hôp., 1897, xiv, 340)	20	26
Edgecombe (Practitioner, London, 1908, lxxx, 194)	5	33
Fabre (Gaz. Méd. de Paris, 1887, iv, 558)	12	58
Jacob (Brit. Med. Jour., 1900, 1, 1532)	1	not stated
Legendre (Bull. Soc. Méd. des Hôpit., 1903, xx, 943)	1	not stated
Lemoine et Lapasset (Bull. Soc. Méd. des Hôp., 1905, xxii, 640)	1	not stated
Pick (Wien. klin. Rundschau, 1902, xvi, 309)	4	20
Priestley (Jour. Amer. Med. Assoc., 1900, ii, 19)	2	not stated
Robson and Cammidge ("The Pancreas, its Surgery and Pathology," Phila. and London, 1907, p. 369)	1	not stated
Simonin (Bull. Soc. Méd. des Hôp., 1903, xx, 928)	10	652
Stevens (Thesis for M. D., Durham, 1901, quoted by Edgecombe, loc. supra cit.)	1	not stated
Zeller (cited by Egdahl, Johns Hopkins Hosp. Bull., 1907, xviii, 130)	1	not stated

In this connection it is interesting to note that *diabetes*, presumably of pancreatic origin, has occasionally followed the mumps (Harris).

Influenza, syphilis, tuberculosis, malaria, typhoid fever, etc., are occasionally accompanied by *chronic pancreatitis,* which is due to the local action of the infecting organism when this circulates in the blood, or, as is probably more often the case, to the toxins produced by the bacteria which are localized elsewhere in the body. Sailer and Speese have succeeded in producing focal necroses in the liver and pancreas of guinea-pigs by injecting blood serum from dogs affected with experimentally induced acute pancreatitis. Injections of normal serum had no such effect. It was also found possible to produce a high degree of immunity by gradually increasing the doses of the toxic serum. Egdahl refers to cases of *acute pancreatitis* following or associated with the following affections: syphilis, typhoid fever, malaria, emboli, furunculosis, bronchitis, heart-disease, pulmonary tuberculosis, and appendicitis. Hirschfeld has quite recently studied the relation of general infections to diseases of the pancreas.

In *arteriosclerosis,* which may be considered an intoxication, if not a toxæmia, the pancreas is quite constantly affected by a chronic indurative inflammation (cirrhosis), and the arteries are often the seat of miliary aneurisms (Lancereaux), a fact which sometimes is held to explain the frequency of hemorrhagic lesions in this organ.

Alcoholism possibly may transmit a non-bacterial inflammation to the pancreas through the blood stream, either directly, or, as is probably much less frequently the case, by chronic passive congestion through hepatic cirrhosis. It is taught by Opie that both hepatic and pancreatic cirrhosis are due to the same cause, and that the pancreatic condition is seldom if ever caused by obstruction of the portal circulation alone. Another condition in which the pancreas is quite constantly involved in a cirrhotic process is that described in 1899 by v. Recklinghausen as *hæmachromatosis,* which in its later stages frequently is accompanied by the *diabète bronzé* of the French.

REFERENCES.

Abrami, Richet, and Saint-Girons: Compt.-Rend., Soc. de Biol., Paris, 1910, lxix, 285, 357.

Desjardins: Thèse de Paris, 1905.

Egdahl: Johns Hopkins Hosp. Bull., 1907, xviii, 130.

Fitz: Bost. Med. and Surg. Jour., 1889, cxx, 181.

Harris: Bost. Med. and Surg. Jour., 1899, cxl, 465.
Hirschfeld: Deutsch. med. Woch., 1909, i, 137.
Lancereaux: Traitè des Malad. du Foie et du Pancréas, Paris, 1899, p. 903.
Macaigne: Bull. Soc. Anat., Paris, 1894, lxix, 366.
Musser: Trans. Pathol. Soc. Phila., 1885, xii, 66.
Opie: Osler's Mod. Med., Phila., 1908, vol. v, p. 652.
Phillips: Lancet, 1908, i, 853.
Roddick: Canada Med. Jour., 1869, v, 385.
Sailer and Speese: Trans. Assoc. Amer. Phys., 1908, xxiii, 540.
Schmackpeffer: Obs. de quibusdam Pancreatis Morbis, Diss. Inaug., Halæ, 1817, f. 25.
Sharp: Brit. Med. Jour., 1908, i, 808.
Simonin: Bull. Soc. Méd. des Hôpit, Paris, 1903, xx, 928.
Tixier and Troisier: Arch de Méd. des Enfants, 1912, xv, 321.

2. *Infection through the Ducts.*—It is usually taught that in the pancreas infection most often occurs by continuity of structure upward from the duodenum, through the papilla of Vater and along the duct of Wirsung. But recent opinions seem to regard this explanation with less favor than was the case a few years ago; for while numerous experiments, which will be mentioned presently, prove that this avenue of infection is quite possible, other experiments and certain anatomical facts tend to make us question its frequent occurrence.

Opie (loc. cit., 1903, page 192) found that the valve-like folds within the diverticulum of Vater prevent the regurgitation of material from the duodenum into the duct of Wirsung, and that if, after death, fluid is forced under considerable pressure into the duodenum, tied above and below the pancreas, none enters the duct. Moreover, in only five out of 223 cases examined did Truhart find bacteria present in the normal duct of Wirsung;[1] and the singular freedom of the duodenum from bacterial life has frequently been pointed out (Vol. I, page 42). As has been seen already (page 5), biliary infection usually occurs primarily through the portal blood stream, being thus a descending infection from the liver; and infection of the ducts, unless these are obstructed by calculi, does not occur to any marked degree because the bile is more or less constantly passing through them, and thus clears them of infectious material. But the gall-bladder, where the

[1] The existence of anaërobic bacteria in the normal biliary and pancreatic ducts has been emphasized as a cause of infection in cases of obstruction by Gilbert and Lippmann.

bile is prone to become a stagnant pool, is frequently the site of infection, and this infection is to all intents and purposes primary there, though really having its origin in the infected bile secreted by the liver. Now in the pancreas there is no such stagnant pool as is found in the gall-bladder; the pancreatic juice is more actively bactericidal than the bile (Remedi); and blood infections of the pancreas are rare; rare also, therefore, is a descending infection of the pancreatic duct.

But there is this to be said in favor of the occurrence of an ascending infection of the pancreatic ducts—that any obstruction to the outflow of pancreatic juice will predispose it to infection, and may make quite possible a pancreatic infection arising in the mildly infectious bile passing through the ampulla of Vater. Such obstruction of the duct of Wirsung frequently occurs in the case of gall-stones impacted in the lower end of the common bile-duct. Besides such factors as these, it must not be overlooked that the accessory pancreatic duct (Santorini) must sometimes be taken into consideration as a possible avenue of infection. It is claimed by Desjardins that, while the normal current of pancreatic juice in the duct of Wirsung flows toward the duodenum, the flow in the duct of Santorini is indifferently either toward the duodenum or away from it toward the intraglandular anastomosis of the two ducts; so that in this way, he asserts, micro-organisms from the duodenum easily and frequently ascend by the duct of Santorini, and meeting the outflowing current in the main duct are carried back through the head of the pancreas, thus doubly infecting the area enclosed between the two ducts. This area has been named by Desjardins the "triangle of infection." This theory takes little account of the supposed bactericidal action of the pancreatic juice, or of the relatively mild infectiousness of the duodenal contents; and fails to explain cases of pancreatic infection in which the duct of Santorini is not patent, or does not anastomose with the duct of Wirsung. Schirmer found the duodenal orifice of the duct of Santorini permeable in only a little over half of the cases (about 100 in number) examined by him. In only forty-eight out of 100 specimens dissected by Opie (loc. cit., 1903, page 30), was the duodenal orifice of the duct of Santorini permeable to injected fluid, while in forty-two cases its duodenal orifice was closed, the duct of Santorini appearing merely as a branch of the main

duct; and in ten cases the two ducts were not in anastomosis at all.

But even if we acknowledge, as it seems we are bound to do, that ascending infection of the pancreatic duct is rare, there nevertheless remain experimental and clinical proofs that infection does occasionally arise in this way. Opie's much quoted case (1901) is an interesting example: at autopsy the cause of acute pancreatitis was found to be a small biliary calculus lodged at the duodenal orifice of the ampulla of Vater; the calculus was so small that it did not obstruct the orifice of the duct of Wirsung, but by occluding the common outlet of this duct and of the common bile-duct, allowed retrojection of bile to occur into the pancreatic duct. Similar results have been obtained experimentally by Carnot, Flexner, and several other investigators (accounts of whose work are given by Flexner), by injecting various substances directly into the duct of Wirsung. Flexner showed subsequently (1906) that when bile is injected into the pancreatic duct, it is the bile-salts which are the destructive agent, and that the more colloid material the bile contains the less intense is the inflammation produced by it. Flexner therefore suggested that, as in chronic inflammation of the biliary passages (as in cholelithiasis) there is a loss of diffusible salts and an increase of colloid material, ascending infection of the pancreatic duct should in such cases lead to chronic rather than acute pancreatitis; and that this is actually the case will be pointed out in a subsequent section. Experimentally chronic pancreatitis has been produced (*a*) by obstruction of the pancreatic duct by a ligature; (*b*) by injecting into the pancreatic duct attenuated cultures of various micro-organisms, modified bile, etc.; (*c*) Carnot produced chronic pancreatitis from an ascending duodenal infection by the ingenious plan of fixing a thread in the duct of Wirsung and carrying it through the ampulla of Vater into the intestine, where it was allowed to hang free. But such experiments as these, as very justly remarked by Maugeret, imply such actual traumatism to the pancreas as to make the interpretation of the results obtained very hazardous. Whether obstruction alone is an efficient factor in producing pancreatitis is a question very difficult to decide, because as soon as the pancreatic secretion becomes stagnated the virulence of the anaërobic bacteria (which,

as already remarked, may be considered normal inhabitants of the ampulla of Vater and lower pancreatic ducts) is markedly increased and the subsequent changes may be attributable largely to their action. As Ebner has tersely expressed it, the results of obstruction are first mechanical, then chemical, and then bacterial.

Intestinal parasites have been observed occasionally in the duct of Wirsung, according to Lieutaud and others. Muroya has recently found ascarides encapsulated in the pancreas.

REFERENCES.

Carnot: Thèse de Paris, 1898, p. 16.
Desjardins: Thèse de Paris, 1905.
Ebner: Volkmann's Samml. klin. Vortr., 1907, Nos. 452 u. 453 (Chir. Nos. 128 u. 129).
Flexner: Johns Hopkins Hosp. Reports, 1900, ix, 743.
Flexner: Jour. of Exper. Med., 1906, viii, 167.
Gilbert and Lippmann: cited by Carnot, "Maladies du Pancréas," Nouv. Traité de Méd. et de Thérap. (Gilbert et Thoinot), Paris, 1908, xx, 107.
Lieutaud: cited by Carnot, "Maladies du Pancréas," Nouv. Traité de Méd. et de Thérap. (Gilbert et Thoinot), Paris, 1908, xx, 109.
Maugeret: Thèse de Paris, 1908, p. 66.
Muroya: Deut. Zeit. f. Chir., 1912, cxix, 21.
Opie: Johns Hopkins Hosp. Bull., 1901, xii, 182.
Opie: Diseases of the Pancreas, Phila., 1903.
Remedi: Arch. Internat. de Chir., 1905–06, ii, 167, 180, 181.
Schirmer: Dissert. Inaug., Basel, 1893, p. 57.
Truhart: cited by Böhm: Beitr. z. klin. Chir., 1904, xliii, 696.

Infection through the Lymphatics.—Largely by exclusion, apparently, investigators are at present led to believe that infection of the pancreas in a majority of cases is due to invasion of its substance by way of the lymph-channels. This view is held by Prof. Thiroloix, and has been developed and ably supported by his pupil Maugeret, who urges that as the efferent lymphatics from the gall-bladder and those from the pancreas anastomose around the head of the pancreas, this part of the gland is in this manner easily invaded directly from the lymph-nodes or lymph-channels. For this pathological state we have adopted the term *pancreatic lymphangeitis*, proposed by Arnsperger. It is a condition which precedes true interstitial pancreatitis, and which is curable by proper surgical treatment. According to this view, which we believe to be

correct for the great majority of cases previously classed together as chronic pancreatitis, it is still the infected bile and the diseased gall-bladder which must be incriminated as the "*fons et origo mali*," though we have also pointed out the possibility of pancreatic lymphangeitis occurring as the result of duodenal and even gastric lesions (page 319). It has also been suggested that though an ascending catarrhal infection along the pancreatic duct is rare, yet micro-organisms may and frequently do travel up *in the walls* of the pancreatic ducts, as they are known to do up the walls of the choledochus, even after this has been ligated. The common bile-duct is imbedded in the head of the pancreas in from 60 to 95 per cent. of cases (Helly, Ebner, Kehr), so that any affection which injures the walls of the choledochus will be very liable to spread to the surrounding pancreatic tissue through the lymphatics. On such grounds has been explained the very frequent occurrence of chronic pancreatitis as a sequel of common duct calculus; but Maugeret contends that this frequent association of pancreatitis with common duct calculus is susceptible of another interpretation, namely, that the calculus is arrested in the common duct by the narrowing of this channel consequent upon the previous existence of a pancreatitis, which in this as in other cases is caused not by a calculus in the common duct, but by infection in the gall-bladder; and she further calls attention to the extreme rarity of such local lesions in the common duct (suppurative or ulcerative angeiocholitis) as could give rise to pancreatitis by contiguity.

The subject of *pancreatic lymphangeitis* is discussed at page 314.

REFERENCES.

Deaver and Pfeiffer: Amer. J. Med. Sc., 1912, i, 473.
Ebner: Volkmann's Samml. klin. Vortr., 1907, Nos. 452 u. 453 (Chir. Nos. 128 u. 129).
Helly: Archiv. f. mikroskop. Anat., 1898, lii, 773.
Kehr: Mitth. a. d. Grenzgeb. d. Med. u. Chir., 1909, xx, 45.
Maugeret: Cholécysto-Pancréatite; Essai de Pathogenie. Thèse de Paris, 1908. Chap. V, *et passim*.

4. *Infection by contiguity* is of infrequent occurrence, unless propagation of infection from the walls of the common duct, which has just been mentioned, is to be included here. The stomach is the organ which is most often at fault in these cases, ulcers per-

forating into, and cancers becoming densely adherent to the pancreas, and setting up a localized inflammatory reaction. Splenic abscess may involve the tail of the pancreas; pyonephrosis, on either side, but usually the left, sometimes has transmitted its infection to the adjacent portion of the pancreas; and the head of the pancreas is not seldom invaded by tuberculosis or other infection localized in the subpyloric or retropancreatic lymph-nodes.

Pancreatic Calculi.—Although concretions in the pancreas were described (de Graaf, 1663) long before cysts (Engel, 1841), pancreatic apoplexy (Spiess, 1866), or fat necrosis (Balser, 1879), they occupy, when compared with biliary calculi, a very insignificant place in the surgery of the pancreas. Opie found only two instances of pancreatic calculi among 1500 autopsies, and thinks that the figures of Giudiceandrea (two in 122 autopsies) much exaggerate their frequency. Lazarus, in 1904, was able to find records of only eighty cases of pancreatic calculi.

FIG. 19.—PANCREATIC CALCULI. (*From a Specimen in the Museum of the German Hospital.*)

Pancreatic calculi occur nearly five times as often in men as in women, according to Lazarus, who found the sex mentioned in fifty-seven cases, forty-seven out of which were men. Lazarus points out that both infection and stasis of the secretion are necessary for the formation of pancreatic concretions. Giudiceandrea found bacteria in the centre of pancreatic calculi, and it is interesting to note that the same experience in regard to salivary calculi, which closely resemble those of the pancreas, has been recorded by Galippe.

Pancreatic calculi usually are multiple, the largest being near the orifice of the main duct, and the others, perhaps hundreds in

number, scattered along its entire length and even in the finest branches of the pancreatic ducts. Occasionally star-shaped or branched calculi are found. The largest stone ever recorded, according to Lazarus, was that of Schupmann, which measured $\frac{1}{2}$ inch by $2\frac{1}{2}$ inches, and weighed, according to Villar, 200 grams. Pancreatic calculi are composed almost entirely of calcium carbonate or calcium phosphate, are not crystalline, and, because of the narrowness of the pancreatic ducts, they are very rarely faceted except at their ends. Usually they are white. Solitary stones, which are quite rare (four among twenty-two cases, Lazarus), are more apt to be composed of oxalates. The nucleus of pancreatic calculi probably is derived from the interaction of the desquamated epithelium lining the ducts with the secretion which has been modified as the result of infection; the result of this interaction is the precipitation of inorganic salts (which are not found in normal pancreatic juice); and new layers are formed as in other calculi (biliary, renal) by the deposit of calcium or magnesium phosphates and carbonates, or even of cholesterin.

While pancreatic calculi are in the first instance the *result* of disease of the pancreas, they become when once formed the *cause* of further structural alterations. By damming up the pancreatic secretion they lead in time to a condition of very marked sclerosis of the gland. Of the eighty cases of pancreatic calculi collected by Lazarus, thirty-six (45 per cent.) had also diabetes, or at least glycosuria.

Carcinoma of the pancreas, however, rarely is associated with the formation of stones. Like calculi of other organs, those of the pancreas occasionally ulcerate their way into other viscera, or even into the peritoneal cavity. Galliard has recorded the case of a patient who was found at autopsy to have a pancreatogastric fistula, one pancreatic calculus lying free in the stomach, and others being still within the pancreas. In Clayton's patient, who died with symptoms of internal hemorrhage, a calculus was found to have ulcerated out of the pancreas into the general peritoneal cavity, opening a blood-vessel; other calculi were found within the ducts of the pancreas.

According to Desjardins, the rarity of pancreatic calculi and the frequency of gall-stones are to be explained by inherent differences between the biliary and the pancreatic systems. He

holds, and Quénu and Duval agree with him, that the same infection arising in the intestine and travelling up the bile- and pancreatic ducts will in the former situation induce a stone-forming catarrh, but in the pancreas will cause a chronic interstitial inflammation.

The symptoms, diagnosis and treatment of pancreatic calculi are considered at page 346.

REFERENCES.

Balser: Arch. f. path. Anat., 1882, xc, 520.
Clayton: London Med. Times, 1849, xx, 37.
Desjardins: Thèse de Paris, 1905.
Engel: Med. Jahrb. d. K. K. osterr. Staates. Wien, 1840, n. F. xxiii, 411; 1841, n. F. xxiv, 193.[1]
Galippe: Semaine Méd., 1893, xiii, 246.
Galliard: Bull. Soc. Anat. Paris, 1880, lv, 191.
deGraaf: "De Succo Pancreatico," London, 1676, p. 81.
Guidiceandrea: Il Policlinico, 1896, iii, Sez. Med., 33; 126.
Lazarus: Zeit. f. klin. Med., 1903-04, li, 530.
Opie: Osler's Modern Med., Phila., 1908, vol. v, p. 657.
Quénu et Duval: Revue de Chir., 1905, xxxii, 401.
Spiess: Schmidt's Jahrb., 1867, cxxxiv, 270.
Villar: xviii Congr. Franç. de Chir., Paris, 1905, p. 495.

Pancreatic Diabetes.—The connection between pancreatic disease and diabetes has been recognized since the time of Cawley (1788), though this observer distinctly stated his opinion that the numerous calculi which were found in the pancreas of his patient were the result, not the cause, of the diabetes. In 1858 Claude Bernard discovered that puncture of the floor of the fourth ventricle caused glycosuria, and for years the nervous theory of the origin of diabetes seemed to rule the medical world. It was not until 1877 that the theory of pancreatic diabetes as a clinical entity was formally propounded by Lancereaux; and the theory was first firmly established as fact by the experimental work of v. Mering and Minkowski, in 1889. Of late years the number of cases of diabetes, in which no lesions are to be found in the pancreas, appears to be steadily decreasing (Bosanquet). This increase in the number of cases of pancreatic diabetes is due largely to more careful examination, especially to microscopical

[1] This work has not been accessible to us.

study of the pancreas post-mortem. According to Cammidge, some observers claim that all cases of diabetes are pancreatic in origin, but this gland has been proved at fault, so far, only in about 88 per cent. of the 288 cases of diabetes mellitus collected by Opie.

The changes in the pancreas which lead to diabetes are believed to be confined to the islands of Langerhans. The secreting parenchyma or the interstitial tissue of the gland may be very extensively diseased, even almost entirely destroyed; yet it seems an established fact that so long as a certain proportion of the islands of Langerhans remains intact, glycosuria does not occur. It is, moreover, apparently proved that the development of glycosuria in disease of the pancreas is to be attributed to interference with the internal secretion of the gland; for it has been found (Minkowski), if a portion of the pancreas is successfully transplanted into the subcutaneous tissues, being severed from all its nervous connections, that the entire portion remaining in the abdomen may be removed and that glycosuria will not develop until the transplanted portion also is removed, or until it becomes atrophic.

It is evident, therefore, as pointed out by Robson and Cammidge, that the cause of glycosuria and of the accumulation of sugar in the blood is dependent upon some influence which the pancreas exerts by way of the blood or lymph stream. Two theories are proposed to explain the means by which these changes are brought about: (1) *The auto-intoxication theory*, by which it is assumed "that the cells of the pancreas normally destroy, or modify, some toxic substance, produced in other parts of the body, which interferes with the utilization of sugar by the tissues;" (2) the theory that the pancreas contributes some *activator substance* to the circulation, which assists a glycolytic enzyme produced by other tissues of the body. The second theory is that which has received most support of late.

The relation of the *adrenals*, and of the *hypophysis cerebri* to pancreatic activity has been too little studied, and is still much too obscure a subject for any authoritative statement to be made at this time. It is perhaps sufficient to recall the cases of pancreatic infantilism referred to at page 259; to note that a diminished function of the anterior part of the hypophysis is

productive of similar changes (von Eiselsberg); that certain cases of acromegaly (*hyperpituitarism*) have been associated with diabetes (Opie); and that the local action of adrenalin upon the pancreas causes temporary glycosuria (Robson and Cammidge); while the external secretion of the pancreas appears to be stimulated by the administration of adrenalin hypodermically (Pemberton and Sweet). Of clinical interest is the case reported by Lavenson, in which symptoms supposed to be due to acute pancreatitis were found at autopsy to have been caused by hemorrhage into the adrenals. Löwi's test for pancreatic disease (mydriasis from instil-

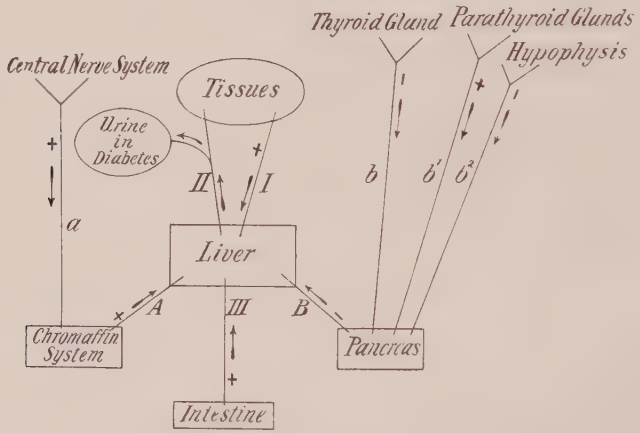

Fig. 20.—Diagram to Explain the Modern Theory of Diabetes. (*von Noorden.*)

lation of adrenalin into the eye), while too recent to be considered authoritative, is another evidence of the little understood relation of the pancreas and the adrenals.

In an interesting study of "The Theory and Treatment of Diabetes," von Noorden has recently endeavored to represent graphically the co-relationship of Pancreas, Liver, Central Nervous System and the Ductless Glands (Fig. 20). He says:

"The manufactory for sugar is the liver, and the liver cells constitute the working department.

"The important claims that *determine* the amount of the sugar production arrive from other organs and tissues, especially from the muscles (path I). The greater the consumption of sugar the stronger will be the claiming impulse, and an amount of sugar sufficient to meet the demand will pass into the blood (path II). This

is normally the only influence which induces the liver to raise its output.

"However, along path III stimuli will also pass. This path represents the blood streaming into the liver from the intestinal wall, and carrying carbohydrates and the products of protein digestion. The quantity of these materials varies according to the type and composition of the food. So long as the manufacture of sugar is well under control the effect of this form of stimulus is not to induce a liberal outpouring of sugar into the general blood stream, but to form glycogen. This glycogen is then stored until it is reconverted into sugar at the request of the tissues, and travels along path II to the muscles, etc.

"In order to maintain the excitability of the sugar manufactories at the proper intensity the two controlling factors come into play. The pancreas, which depresses the excitability, sends its secretion along path B, while the suprarenals, which increase the excitability, distribute their secretion along path A. This conjoined action serves to keep the process in equilibrium.

"Both these controlling glands are in turn influenced by other organs—the pancreas through the thyroid gland path (b). and the suprarenal through the nerve paths (a). The diagram shows still other influences (paths b^1 and b^2); their existence is most probable, but their origin is undetermined. They seem to be subordinate in importance and action."

REFERENCES.

Bosanquet: Lancet, 1905, i, 903.
Cammidge: Surg., Gyn., and Obstet., 1908, vi, 22.
Cawley: London Med. Jour., 1788, ix, 286.
v. Eiselsberg: Annals of Surg., 1910, ii, 1.
Lancereaux: Bull. de l'Acad. de Méd., Paris, 1877, 2e, Série, vi, 1215.
Lavenson: Arch. of Internal Med., 1908, ii, 62.
Löwi: Arch. f. exp. Path. u. Pharm., 1908, lix, 83.
v. Mering and Minkowski: Centralbl. f. klin. Med., 1889, x, 393, Arch. f. exp. Path. u. Therap. 1889, xxvi, 371.
von Noorden: Amer. Jour. Med. Sc., 1913, i, 1.
Opie: Osler's Modern Med., Phila., 1908, v, 627.
Pemberton and Sweet: Trans. Coll. Phys. Phila., 1910, xxxii, 176.
Robson and Cammidge: The Pancreas, its Surgery and Pathology, Phila., 1907, p. 273; 276.

General Diagnostic Considerations.

Though disease of the pancreas is now recognized as not very rare, and the contention of Robson, that its diagnosis usually is possible, is very generally accepted as true; it is nevertheless a fact, owing to the deep situation of the pancreas, and its close anatomical and physiological relations with surrounding organs, that such diagnosis frequently is difficult, and that pancreatic lesions often escape even acute observation.

The pancreas has both *digestive* and *metabolic* functions, the disturbance of which by disease causes more or less definite symptoms; and there are certain *physical signs* which usually may be elicited in cases of pancreatic disease.

Digestive Symptoms.—It is well known that the digestive functions of the pancreas may be partially assumed by other organs. Possibly if the pancreas could be removed piecemeal, destroying its functions one by one in very gradual stages, the loss of its secretions might be entirely supplied by compensatory action of the salivary glands, the gastric and the intestinal juices. The amylolytic function of the pancreas is supplemented by the saliva; the tryptic function is the same as that of the pepsin of the gastric juice; and the intestinal juices and bacteria, aided by the bile, are capable of caring for a large part of the ingested fat. But when disease attacks the pancreas, the destruction of its physiological functions often takes place so suddenly that very pronounced and definite symptoms are produced. Chief among these are *steatorrhœa*, and *azotorrhœa*; others are *sialorrhœa*, *dyspepsia* (a vague term), *emaciation*, *nausea*, *vomiting*, etc. The administration of pancreatic extract, pancreatin, etc., as a means of obviating these symptoms, has been used as a diagnostic test by Salomon and others.

Steatorrhœa.—An excess of fat in the fæces was first recognized in 1820 by Kuntzmann as due to pancreatic disease. In health the fæces normally contain about 20 to 21 per cent. of fat (Fitz, Robson and Cammidge), which represents from 7 to 11 per cent. of the fat taken as food. In pancreatic disease the total amount of unabsorbed fat may reach 50 to 60 per cent. in cases of chronic pancreatitis, and even 75 to 90 per cent. in cases of malignant disease (Robson and Cammidge). Carnot says that after suppression of

the bile alone the fæces contain 60 per cent. of fat; after suppression of the pancreatic secretion, 70 per cent. of fat; and after suppression of both bile and pancreatic juice, they contain 90 per cent. of fat. In health, according to Müller, "the unabsorbed fæcal fats consist of approximately 20 to 30 per cent. of neutral fat and from 70 to 80 per cent. of split fat, which is partly fatty acids and partly soaps." R. Gaultier has shown that in cases of suppression of the bile there are found in the fæces from 35 to 45 per cent. of the ingested fats: 63 per cent. of this exists as neutral fats, and 35 to 40 per cent. as split fats (21 per cent. as fatty acids, and 12 per cent. as soaps); he has found that in cases of suppression of the pancreatic functions 80 per cent. of the fæcal fat is in the form of neutral fats, and only about 15 per cent. as split fats (10 per cent. as fatty acids, and 5 per cent. as soaps); and if both biliary and pancreatic secretions are absent, 90 per cent. of the ingested fat is recovered from the fæces, of which 90 per cent. is in the form of neutral fats, and 8 per cent. as split fats (7 per cent. as fatty acids, and 1 per cent. as soaps.) Thus where both bile and pancreatic juice are lacking, the ingested fat is used hardly at all, and the split fats found in the excreta are reduced at least to one-fifth of normal. Müller found in cases of pancreatic disease that even though the total percentage of fæcal fat might not be increased above the normal, yet that the porportion of split fat is always decreased (averaging about 40 per cent. of the total fat), showing decrease in the digestive power for fatty food. It has been claimed by Katz that diminution of the split fat below 70 per cent. of the total fæcal fat invariably signifies disease of the pancreas, except in nursing infants and patients with profuse diarrhœa.

The excess of the fat in the fæces may be evident to the most casual observation, or may be demonstrable only by the microscope. In well-marked cases of steatorrhœa the passages are bulky, of a silver, gray, or asbestos-like color; and the fat may float on the surface of the fluid mass like oil droplets or particles of butter. Such passages have been known occasionally to occur in health, after the ingestion of abnormal quantities of fatty food, as individuals differ in their ability to digest fats; and diminution of the secretion of bile, diarrhœa, caseation of the mesenteric lymph-nodes, and other intestinal derangements, as well as pancreatic disease, may give rise to this symptom.

Azotorrhœa.—The presence in the fæces of undigested proteid material was first recognized as a symptom of pancreatic disease by Fles, in 1864. In health, according to Opie, about 5 or 6 per cent. of the nitrogen ingested with the food is excreted in the fæces; whereas in cases of proved or supposed pancreatic disease various investigators have recovered from the fæces as much as 32, 45 and even 70 per cent. of the ingested nitrogen. Azotorrhœa is a less constant, less valuable, and less easily observed symptom than steatorrhœa. Fitz has pointed out that the condition occurs "only when there is extreme diminution of the pancreatic juice, and is significant only when gastric digestion is normal, when the diet contains no excess of meat, and when there is no diarrhœa" (Opie). If gastric digestion be deficient, the meat fibres will not be separated from each other, and the trypsin of the pancreatic juice will be able to act only on the surface of the meat bundles.

Schmidt observed that a safer test than mere digestion of the meat fibres was the *destruction of the nuclei of the muscle cells*, since these are digested only by the pancreatic juice; his test consists in feeding to patients, suspected of having pancreatic disease, small cubes of beef enclosed in little silk bags; these bags are readily recovered from the fæces, and their contents are examined microscopically to determine whether by penetration of pancreatic juice the muscle-cell nuclei have been destroyed.

Müller's test, to determine the presence of a proteolytic ferment (trypsin) in the fæces, is described by Lyle: after the administration of a test-meal, and suitable preparation of the fæcal mass, small drops of the latter are plated on a Petri dish of coagulated blood serum, and this is incubated; if a proteolytic ferment is present, small depressions are produced in the blood serum wherever the fæcal drops have been placed.

Sahli's test for the efficiency of proteid digestion depends on the fact that gelatin capsules hardened in formol resist gastric digestion for twelve hours or more, but are rapidly digested by pancreatic juice. By administering such capsules filled with iodoform the fact of their disintegration can be proved by finding iodine in the urine or saliva; the reaction appears in health in from four to eight hours, and if there is no marked loss of gastric motility a delayed reaction indicates impairment of pancreatic function. The test

is by no means accurate, but may sometimes aid in making a diagnosis.

Salomon's test, which is commended by Ochsner, depends on the fact noted in 1898 by Deucher, that lecithin is present in unusual quantities in the fæces of patients with pancreatic disease. Salomon found that in patients with pancreatic disease on an egg diet from 0.4 to 1.2 grammes of lecithin are excreted in the fæces daily; whereas if the pancreas is normal, never more than 0.1 gramme is excreted. He states that in uncomplicated cases of biliary obstruction Jürgensen found the quantity varied from 0.1 to 0.4 grammes *pro die*. Salomon also claims that patients with pancreatic disease who are fed on v. Noorden's oatmeal diet have typical "butter stools."

Carbohydrate Digestion.—Fedeli and Romanelli have recently described a test for the determination of the functional activity of the pancreas, which they found was quite as accurate as Müller's test: their test is based upon the fact, proved by Roger and Simon, that saliva is inhibited by the gastric juice but reactivated by the pancreatic juice if in an alkaline medium. By mixing a certain portion of the patient's saliva with gastric juice (or HCl) and an indicator (carbohydrate), and then rendering the mixture alkaline, the presence of pancreatic ferment in the fæces is shown by its reactivating effect on the saliva, which digests the carbohydrate indicator when fæces and saliva are mixed.

The presence of **stercobilin in the fæces** indicates that there is not complete obstruction to the discharge of bile into the intestines. Robson and Cammidge have found that it is entirely absent or present in only very faint traces in cases of carcinoma of the head or the pancreas, where biliary obstruction usually is complete at the time these patients come under the surgeon's observation; whereas in cases of obstructive jaundice due to other causes (chronic pancreatitis, common duct cholelithiasis) the obstruction is rarely absolute, and a distinct though often subnormal reaction for stercobilin may be obtained.

Other matters in the patient's stools may call attention to the pancreas as the seat of disease. While there is nothing pathognomonic about *blood* or *pus* in the motions, yet the periodic discharge of *saliva-like fluid*, which has been noted in some cases of pancreatic cysts, or the passage of pancreatic calculi, could

scarcely fail to arrest the attention of both patient and surgeon. Chiari's and Trafeyer's patients passed a *gangrenous pancreas* by rectum.

Sialorrhœa Pancreatica.—Under this name is described an increased flow of saliva which has been observed in a few cases of pancreatic disease, and which is regarded by Robson and Cammidge as significant of efforts at compensation; in one of their patients troublesome salivation "ceased in a most striking manner within forty-eight hours after he had been operated on for chronic pancreatitis." Carnot refers to other cases recorded by Battersby, Ludolph, Holzmann, Capparelli, and Guidiceandrea.

Dyspepsia due to pancreatic disease is difficult to differentiate from those forms caused by biliary or gastric affections. Anorexia is more marked in gastric affections. Loss of weight in spite of ingestion of nearly normal amounts of food frequently characterizes pancreatic disease. Pain is less acute than in gastric dyspepsia, and attacks of colic much less usual than in gall-bladder disease.

Emaciation, as already noted, is often a prominent and highly suggestive symptom of disease of the pancreas. The loss of flesh is rapid, persistent, and very generally observed by both patient and physician.

Metabolic Symptoms. Glycosuria.—As was pointed out in the section on pancreatic diabetes, this condition is associated with change in the islands of Langerhans, and occurs only when their destruction is very widespread; moreover, pancreatic diabetes may be unaccompanied by any symptoms of impairment of the external secretion of the pancreas. Glycosuria, therefore, is not a frequent symptom of such pancreatic lesions as have interest for the surgeon; it usually indicates a very advanced lesion, which, according to Opie, Robson and Cammidge, and other observers, is most frequently chronic interstitial pancreatitis of the interlobular type. Interacinar pancreatitis, though producing glycosuria much sooner, is a much rarer affection.

Dextrose is the sugar usually found in the urine of pancreatic diabetes, but maltose and even pentose occasionally are present.

The "*pancreatic reaction*" of Cammidge consisting in the crystallization of an unknown substance, possibly a pentose, from the urine of patients with pancreatic disease, was mentioned

by Robson in 1901, in a paper read before the American Surgical Association. The methods of making this test have been variously modified and improved by Cammidge. As succinctly stated by Opie, the test consists in applying the phenylhydrazine test for sugar to urine previously boiled with strong hydrochloric acid and treated with basic lead acetate, in order to remove glycuronic acid; in the case of pancreatic disease sheaves of yellow crystals are obtained, which are soluble in dilute sulphuric acid. Robson and Cammidge state (1907) that in their experience (200 cases) "a characteristic pancreatic reaction in the urine has always been associated with evidence of disease of the pancreas at operation, or post-mortem, in all cases where it has been possible to investigate the condition of the gland." Many theoretical objections have been made to the supposed value of this test of Cammidge, by investigators in this country, in Great Britain, and on the continent. (References may be found in the article of Schmidt, cited at page 288.) The most weighty objection, it seems to us, is that the value of the test depends so largely on the individual making the test, though the personal equation is not so important with the "improved test" as it was when the earlier tests were employed. Some of those who have most practical experience with the "improved test" (Schmidt, Kehr, Goodman, Watson) speak favorably of its diagnostic value, though it is sometimes present in other abdominal diseases. In our own practice we have gradually come to regard it as of less and less value. Kinney has made two series of examinations for the Cammidge reaction in patients under the care of the senior author at the German Hospital. These series (154 tests in the first, and 197 in the second) comprise 351 tests in all, constituting a very fair experience by which to judge the test. Roughly speaking, in all the cases in which the condition of the pancreas was determined accurately at the time of operation, this supposedly specific pancreatic reaction was obtained only about two and a half times as frequently when the pancreas was affected as when it was not.

Alimentary Glycosuria.—Even though the diet of a healthy individual contains a great excess of sugar, it is only very exceptionally that glycosuria occurs during the process of digestion. But this alimentary glycosuria is much more apt to occur, and may be a constant phenomenon, if there is serious disease of the pan-

creas. It has been found by Wille, after whom the test frequently is named, that in about 65 per cent. of cases of alimentary glycosuria there is serious disease of the pancreas. Dextrose (fruit sugar) is better for the test than cane sugar: 100 grams of dextrose are given in a half pint of water on the fasting stomach, and the urine is examined two or three hours later.

Opie succeeded in one case in demonstrating the presence of a *fat-splitting ferment in the urine;* and Hewlett found a similar ferment in the urine of dogs for a period of from three to five days after experimental injuries of the pancreas.

Wohlgemuth and Noguchi found in experiments on dogs, after contusion of the pancreas, that *diastase was found in both blood and urine.* They think the test should be even more accurate in man than in the dog, and that it should appear promptly in every case of pancreatic lesion.

Physical Signs.—Inspection may show *emaciation*, which, if of rapid occurrence, in connection with digestive symptoms, is always suggestive of pancreatic disease. *Jaundice* is another physical sign very evident when it occurs, but by no means so frequent in pancreatic disease as the relation of biliary lesions to the latter might lead one to expect. The occurrence of jaundice depends upon the relation of the common bile-duct to the head of the pancreas; and Robson and Cammidge call attention to the coincidence of their figures with Helly's anatomical investigations: as was stated at page 267, Helly found the common duct embedded in pancreatic tissue in 62 per cent. of cases; and Robson and Cammidge found bile-pigments in the urine of 62 per cent. of those cases of chronic pancreatitis which were associated with cholelithiasis. The induration of the pancreas may be sufficient to obstruct the outflow of bile even if no gall-stones are present. Robson and Cammidge observed jaundice in 16 per cent. of such cases. Robson, Kehr, and others think that many cases of so-called "catarrhal jaundice" are due to pancreatitis of mild degree. Steadily increasing, painless jaundice, in time becoming almost black, with distention of the gall-bladder, is very characteristic of carcinoma of the head of the pancreas, or of the papilla of Vater.

Fat necrosis, first studied by Balser in 1882, may be seen when the abdomen has been opened; it is due, probably with very few

exceptions, to pancreatic disease.[1] By fat necrosis is meant the result of the action of the steapsin of the pancreatic juice upon surrounding fat areas, resulting in the splitting of this fat into acids and glycerine. "Fatty acids," says Opie, "are deposited as needle-like crystals within the cell, which has lost its nucleus and is evidently necrotic, while the soluble glycerine is absorbed. Very soon the fatty acids unite with calcium, to form calcium salts; and within the cell outline which is still preserved are irregular, often globular masses, in which the presence of lime salts may be demonstrated by micro-chemical reactions." According to Ebner fat necrosis may occur (1) *directly by trauma*, permitting the access of pancreatic secretion to the tissues immediately surrounding; (2) *through the lymphatics*, as noted by Bryant, Böhm, and Gulecke; (3) *through the blood*, for although Tschepurowski found that the action of steapsin is inhibited in the circulating blood, yet Payr and Martina have found emboli composed of pancreatic cells. Fat necrosis which is found widely disseminated in the abdomen (omentum, mesentery) is more apt to be due to diffusion of steapsin through lymphatic spaces than to direct transperitoneal contact of the pancreatic juice; and certainly in the rarer instances of fat necrosis of the subperitoneal, subcutaneous, and pericardial fat, any direct transperitoneal access of the pancreatic secretion is out of the question. Bunge explained the occasional absence of fat necrosis in suppurative pancreatitis by rapid thrombosis of the lymph-stream.

The areas of fat necrosis vary in size from less than one to more than five millimetres, usually being visible as minute whitish specks or flakes, of dense rigid feel, often surrounded by a hemorrhagic zone, and not raised from the surface of the surrounding fat, a fact which aids in distinguishing them from miliary tubercles. It is not unlikely that very minute areas of fat necrosis often are overlooked. The whitish specks may be made more conspicuous, as Bender has shown, by the application of a half-saturated solution of copper acetate, which turns the affected area green, showing a fine contrast from the yellow of the normal surrounding fat.

Fat necrosis occurs in the acute lesions of the pancreas (infections and traumatisms) with much greater frequency than in

[1] Richter notes its presence in a case of perforated duodenal ulcer.

chronic affections (interstitial pancreatitis, carcinoma), but it appears to have been definitely established that it is not itself caused by bacteria; these are a mere coincidence. In one case recorded by Hansemann, areas of fat necrosis in the subcutaneous tissue were found to correspond with circumscribed hemorrhagic areas visible through the skin.

Hemorrhage.—Another characteristic of disease of the pancreas is a hemorrhagic tendency. On opening the abdomen in acute cases there may be found a bloody exudate bathing the entire abdomen; various discrete hæmatomata, sometimes widely distinct from the pancreas, may be encountered; and frequently the pancreas itself is the site of a massive hæmatoma, or diffuse petechial hemorrhages. It was pointed out by Körte, as long ago as 1894, that these hæmatomata were not scattered around by chance, but that their situation might be explained on anatomical grounds, owing to the disposition of the various "peritoneal leaves" or "fascias d' accolement" around the pancreas. Leriche and Arnaud claim that the sanguinolent effusion in the general peritoneal cavity is due to rupture of the gastro-colic omentum (either by trauma or by the digestive action of the pancreatic ferments), or leakage of pancreatic juice through the foramen of Winslow, or the escape somewhere of the bloody exudate arising originally in the pancreas.

After operation on patients with pancreatic disease, especially infections and carcinoma, there is not seldom manifested a tendency to hemorrhage, not confined to the wound alone; oozing may occur from previously intact mucous membranes, and subcutaneous bleeding, causing disfiguring ecchymoses, may result from trifling causes. Truhart and Doberauer accuse the digestive action of the pancreatic trypsin on the neighboring tissues, opening venules and arterioles, as the cause of this hemorrhagic tendency; while poverty of the blood in lime salts, which are excreted in excess in cases of pancreatic disease, is thought by Robson and Cammidge to be a sufficient explanation.

Œdema of the lower extremities, from pressure on the inferior vena cava, is a rare sign of pancreatic disease, as is *ascites* from pressure on the portal vein. The senior author has met with ascites in one case.

Palpation.—In emaciated patients, and especially in those

with gastroptosis, the diseased pancreas often can be detected by palpation as an oblong tumor, lying transversely above the umbilicus, of firm consistency, and usually more cord-like than might be expected. Carcinoma of the head of the pancreas could be more often detected by palpation, were it not obscured by a distended gall-bladder, one of the most characteristic physical signs of this lesion. In cases of subacute and suppurative pancreatitis it is almost always possible to detect a deeply-seated diffuse resistance in the epigastrium, which on opening the abdomen is found to have been caused by the pancreas, engorged with bloody and inflammatory exudate, or with pus. In cases of acute pancreatitis rigidity of the belly wall may prevent satisfactory palpation. Cysts of the pancreas almost always can be felt on palpation, but their differential diagnosis is more easily made by percussion, as will be presently described. Hydronephrosis, which in a few cases (Chvostek, Boldt) has resulted from pressure by the pancreas on the ureter, usually can be recognized by palpation. On the other hand, in a case reported by Villard and Thévenet, a calculous hydronephrosis simulated a tumor of the pancreas because it produced obstructive jaundice. Extravasations in cases of acute pancreatitis sometimes are more easily discovered by palpation in the left loin than in the epigastric region.

Percussion.—This is of most value in determining the relation of the stomach and colon to supposed cysts or tumors of the pancreas. By distention of these viscera with air, by means of a hand-bulb attached to stomach or rectal tube, it usually is possible to ascertain (1) that a given tumor is retroperitoneal in origin; (2) that it presents (a) through the gastro-hepatic omentum, (b) through the gastro-colic omentum, or (c) below the transverse colon. By percussion, also, the size of the liver may be determined; when this is much enlarged, and is accompanied by a distended gall-bladder and deep jaundice, the diagnosis of carcinoma of the pancreas is very probable (Fig. 27).

Diagnosis.—In attempting to make a diagnosis in a case of suspected pancreatic lesion, the surgeon should consider: (1) The predisposing causes; (2) the clinical history; (3) symptoms; (4) physical examination; and (5) the results of the laboratory tests of urine, fæces, etc.

1. **Predisposing Causes.**—Under this heading usually are considered sex, age, race, etc.

Sex.—The male sex undoubtedly is more predisposed to pancreatic disease than the female; according to Böhm, large statistics show that 65 per cent. of cases of pancreatic disease occur in men.

Age.—Most patients with disease of the pancreas are of middle or later life; in young men or women it is said to occur chiefly in hard drinkers, prematurely aged.

Race.—We are not aware that marked prevalence of pancreatic disease in any race has been observed; but in our own experience members of the Hebrew race seem to have been in the majority, as is also the case in disease of the biliary tract (page 89).

Habits.—It has long been taught that hard drinkers and high livers are exceptionally predisposed to pancreatic lesions. This has not been our own experience; we have found, however, that many of our patients with pancreatitis (acute or chronic) had arteriosclerosis from one cause or another.

Obesity is another factor which is looked upon by some as a predisposing factor of consequence.

Certain other diseases, of which arteriosclerosis has already been mentioned, undoubtedly greatly predispose to the development of pancreatic lesions. By far the most important of these is *biliary infection:* Quénu and Duval in 1905 computed that gall-stones were present in 50 per cent. of cases of pancreatitis; Egdahl says about 42 per cent. of cases of pancreatitis are associated with gall-stones; Kehr found evidence of chronic pancreatitis in sixty-nine (30 per cent.) of his last 220 operations for biliary disease; Mayo has found 81 per cent. of his cases of pancreatic disease caused by or coincident with cholelithiasis. As Kehr points out, the higher percentage of cases of pancreatic disease reported in recent statistics is to be explained by more careful examination of the pancreas at operation, such investigation being indicated whenever the Cammidge reaction is positive. Before making use of this test Kehr found pancreatic disease in only 10 per cent. of his cases of cholelithiasis. Of seventy-nine patients with chronic pancreatitis under the senior author's care in the German Hospital, seventy-two (91 per cent.) showed evidence of infection of the bile-passages; forty-two (53 per cent.) had gall-stones, and

in thirty (38 per cent.) there was non-calculous inflammation. Statistics may also be given of the percentage of cases of gall-stone disease in which lesions of the pancreas are noted; thus Mayo found that only 359 (8.9 per cent.) of his 4000 cases of biliary disease had also noticeable lesions of the pancreas; Kehr found that in 24 per cent. of his patients with disease of the biliary tract palpable lesions of the pancreas were present. Perhaps it is just as well in this place to note that a positive diagnosis of chronic pancreatitis cannot always be made merely from palpation of the organ during an operation. Many a time such a diagnosis has been made, and when opportunity has offered later to test the diagnosis by the use of the microscope no lesions have been found. These probably are cases of pancreatic lymphangeitis. The influence of *general infections* in producing lesions of the pancreas has already been noted (page 262). Finally *injury* should be remembered as a predisposing cause of considerable importance (page 351).

2. **History.**—A good clinical history of the patient is of the utmost importance. The previous existence of general infections, especially of typhoid fever, owing to its predilection for the biliary tract, the occurrence of jaundice, dyspepsia, colic, or "stomach cramps," any one of these factors should bring to mind the possibility of pancreatic infection as a sequel.

3. **Symptoms.**—No matter how trivial at first glance, all symptoms should be cautiously weighed, as possibly having some bearing on the diagnosis of pancreatic disease. Thus dyspnœa, which many will think can have no special diagnostic value, is dwelt upon by both Riedel and Musser, as especially characteristic of pancreatic disease; the former states that when during an attack of supposed biliary colic dyspnœa is a marked symptom the surgeon always should think of pancreatitis as a complication; Musser also notes it as a symptom characteristic of acute pancreatitis. Dyspnœa is thought to be produced either by reflex nervous action through pressure on the solar plexus, or by means of toxic matters in the circulation. Collapse, which often is profound in cases of acute pancreatitis, is attributed to similar causes. Cyanosis is a not unusual accompaniment of collapse and dyspnœa. Pain, in acute cases, is excruciating; in chronic pancreatitis and in cancer of the pancreas it usually is not very severe, unless perigastric adhesions

exist, or unless neighboring nerve trunks are compressed. Pain is often referred to the left shoulder blade, or to the middle of the back between the shoulders. Epigastric pain may be increased after taking food, possibly, as suggested by Martina, because the physiological activity of the pancreas some hours after a meal causes the gland to swell up and distend its peritoneal capsule. A very severe pain may be caused by small, localized, intrapancreatic hemorrhages. Rapidity of the pulse may indicate the hemorrhagic nature of the pancreatic lesion. Fever usually exists in cases of infection of the pancreas, though in cases of pancreatic apoplexy and other hyperacute lesions where collapse occurs the temperature may be subnormal.

4. **Physical Examination.**—This should be systematic and complete. *Inspection* shows the presence or absence of emaciation, jaundice, tumor, dyspnœa, cyanosis, etc. *Palpation* may detect rigidity of the abdominal walls; tenderness to pressure in the epigastrium; a deeply placed sense of resistance or a well-defined tumor; fulness in the left flank; a distended gall-bladder, etc. *Percussion* enables the surgeon to determine the relation of the stomach and colon to any suspected pancreatic swelling; and to outline the liver and gall-bladder.

5. **Laboratory Tests.**—Many of these have already been mentioned. The most important are, for *examination of the fæces:* 1. Excess of fat, and diminution in the proportion of split fat contained in the fæces. 2. Presence of undigested proteid material, as determined by Schmidt's test (page 276). 3. Absence of proteolytic ferment as shown by the test of Fedeli and Romanelli (page 277). In the *examination of urine*, the most valuable tests are: 1. The pancreatic reaction of Cammidge (page 278). 2. Alimentary glycosuria (Wille's test, page 279). For detailed descriptions of these various tests the reader is referred to the original communications of the authors, references to which are appended; and especially to the monograph of R. Gaultier, and the paper by Terrier, cited below.

REFERENCES.

Balser: Arch. f. path. Anat., 1882, xc, 520.
Bender: cited by Robson and Cammidge, loc. cit., p. 194.
Böhm: Beitr. z. klin. Chir., 1904, xliii, 694.

Boldt: quoted by Martina, loc. cit.
Bunge: quoted by Ebner, loc. cit.
Cammidge: Lancet, 1904, i, 782.
Cammidge: Brit. Med. Jour., 1906, i, 1150.
Carnot: Mal. du Pancréas. Nouv. Traité de Méd. et de Thérap. (Gilbert et Thoinot), Paris, 1908, xx, 141.
Chvostek: quoted by Martina, loc. cit.
Deaver: Jour. Amer. Med. Assoc., 1911, i, 1079.
Doberauer: quoted by Carnot, loc. cit.
Ebner: Volkmann's Samml. klin. Vortr., 1907, Nos. 452 u. 453 (Chir. Nos. 128 u. 129).
Egdahl: Johns Hopkins Hosp. Bull., 1907, xviii, 130.
Fedeli e Romanelli: Riforma Medica, 1909, xxv, 1043.
Fitz: Trans. Congr. Amer. Phys. and Surg., 1903, vi, 41.
Fitz: quoted by Opie, loc. cit., p. 624. (Azotorrhœa).
Fles: Holland. Arch., 1864, ii, 187, cited by Robson and Cammidge, loc. cit., p. 344.
Gaultier: De l' Exploration de l' Intestin per l' analyse des Fèces. Paris, 1905.
Goodman: Annals of Surg., 1909, i, 183.
Haggard: Jour. Amer. Med. Assoc., 1908, ii, 377.
Hansemann: Berl. klin. Woch., 1889, cited by Robson and Cammidge, loc cit., p. 194.
Hewlett: Jour. of Med. Research, 1904, n. s., vi, 377.
Katz: Wien. med. Woch., 1899, xlix, 153, 214, 266.
Körte: Verhandl. d. Deutsch. Gesellsch. f. Chir., 1894, xxiii, ii, 373.
Kehr: Mitth. a. d. Grenzgeb. d. Med. u. Chir., 1909, xx, 45.
Kinney: Amer. J. Med. Sc., 1910, ii, 878.
Kuntzmann: Jour. d. pract. Arzneykunde u. Wundarzneykunst. (Hufeland), Berlin, 1821, liii (n. s. xxxxvi), ii, 106.
Leriche and Arnaud: Rev. de Gyn. et de Chir. Abd., 1909, xiii, 659; 795.
Lyle: N. Y. Med. Jour., 1910, i, 1103.
Martina: Deutsch. Zeit. f. Chir., 1907, lxxxvii, 499, 525.
Mayo: Collected Papers (1911) Phila. and N. Y., 1912, p. 151.
Mayo: Surg. Gynec. and Obst., 1908, vii, 607.
Mayo: Jour. Amer. Med. Assoc., 1908, i, 1161.
Müller: cited by Opie, Osler's Mod. Med., Phila., 1908, v, 622.
Müller u. Schlecht: Med. Klinik, 1909, v, 573.
Musser: Trans. Assoc., Amer. Phys., 1908, xxiii, 555.
Ochsner: Surg., Gynec., and Obst., 1908, vii, 621.
Opie: Osler's Modern Medicine, Phila., 1908, v. 621.
Quénu et Duval: Rev. de Chir., 1905, xxxii, 401.
Richter: Quarterly Bull. Northwestern Univ. Med. School, Chicago, 1910.[1]
Riedel: Die Path., Diag., u. Behandl. des Gallensteinleidens. Jena, 1903, S. 36.

[1] This work has not been accessible to us.

Robson: Trans. Amer. Surg. Assoc., 1901, xix, 151.
Robson and Cammidge: The Pancreas, its Surg. and Pathol., Phila., 1907.
Sahli: Berl. klin. Woch., 1902, xxxix, 249.
Sahli: Deutsch. Arch. f. klin. Med., 1898, lxi, 445.
Salomon: Wien. klin. Woch., 1908, xxi, 480.
Schmidt: Die Funktionsprüfung des Darmes, Wiesbaden, 1908, S. 49.
Schmidt: Test Diet in Intestinal Dis., Trans. by C. D. Aaron, Phila., 1909, p. 73.
Schmidt: Mitth. a. d. Grenzgeb. d. Med. u. Chir., 1909, xx, 426.
Terrier: Bull. et Mém. Soc. Chir., Paris, 1906, xxxii, 164.
Trafeyer: cited by Chiari: Wien. med. Woch., 1880, xxx, 139.
Truhart: quoted by Carnot, loc. cit., p. 195.
Villard and Thévenet: Lyon Méd., 1909, cxiii, 27.
Watson: Brit. Med. Jour., 1908, i, 858.
Williams and Busch: Trans. Assoc. Amer. Phys., 1907, xxii, 304.
Wille: Deutsch. Arch. f. klin. Med., 1899, lxiii, 546.

Acute Pancreatitis.

Under this category are included by Mayo Robson both catarrhal and parenchymatous inflammations. "The former," he says, "resemble the different forms of cholangeitis, with which indeed they are frequently associated; the latter bear more resemblance to inflammatory affections of the appendix, 'suppurative' and 'gangrenous' appendicitis." The acute catarrhal inflammations of the pancreas are neither so frequent nor so important in surgery as the parenchymatous inflammations. They are attended by moderate swelling of the head of the gland, which may compress the common duct and thus give rise to "catarrhal jaundice," which, as previously mentioned (page 280), often may be due to such a condition as this rather than to œdema of the mucous membrane of the bile-duct or occlusion of its duodenal orifice. Suppurative catarrh of the pancreatic ducts has also been observed. But much more importance attaches to pancreatic lymphangeitis and to chronic catarrhal pancreatitis, which, as will be shown subsequently, are frequent forerunners of chronic interstitial pancreatitis; the pathogenesis, symptoms, and treatment of these conditions are discussed in connection with the latter subject (page 314).

The parenchymatous forms of acute pancreatitis have been classified by Fitz (1889) as *hemorrhagic*, *suppurative*, and *gangren-*

ous. The differences are those of degree, rather than of kind; and while gangrenous pancreatitis nearly invariably, and suppurative pancreatitis frequently, is a sequel of the primary hemorrhagic change, either form may arise independently, though the gangrenous rarely does so. The fullest account will be accorded the primary, hemorrhagic form.

Hemorrhagic Pancreatitis.

The tendency to hemorrhage in disease of the pancreas has already been discussed; but it must not be thought that the use of the term hemorrhagic pancreatitis implies any form of inflammation peculiar to the pancreas alone. It is well known that in other structures a hemorrhagic form of inflammation is not seldom observed; and the more that is learned of pathological processes in general, and of the special pathology of the pancreas in particular, the more evident does it become that the pancreas conforms to general pathological laws.

Pathogenesis.—Hemorrhagic pancreatitis has been produced experimentally (Carnot) by trauma, by injecting chemicals into the duct of Wirsung or directly into the parenchyma of the gland, and perhaps most interesting of all by injections of bile, gastric juice, and even normal pancreatic juice, trypsin, etc. In connection with this auto-digestive action of the pancreatic secretion, the important point to observe, as explained in Vol. I, page 38, is that the pancreatic juice is activated by a kinase with which it comes in contact only after leaving the pancreas. Now, it has been pointed out by Carnot that, under abnormal conditions, a kinase generated by leukocytes or even by bacteria, within the pancreas, can activate trypsinogen and convert it into trypsin, and that if this is produced within the pancreas it will have the power of digesting the surrounding proteid material. By a similar pathological law, the pepsin of the gastric juice is activated only by hydrochloric acid; and as it is held by some that auto-digestion of the stomach, in certain cases, may produce "round ulcer" in that organ, so from analogy it is suggested (Truhart) that trypsin may be the direct cause of intrapancreatic hemorrhages, producing veritable "round ulcers" of the pancreas (Desjardins). The pancreas, as is well known, is extremely susceptible to *post-mortem* auto-

digestion; indeed it has been claimed by Chiari that this process begins in approximately 50 per cent. of cadavers within a few hours of death, or even during the agonal period; and he has expressed the opinion "that idiopathic hemorrhagic or gangrenous pancreatitis for the most part is nothing other than an intravital tryptic auto-digestion of the pancreas"(Williams and Busch).

On the other hand, the toxic agent producing the hemorrhage may be held to circulate in the blood or lymph streams, as in the toxæmic theory of the origin of gastric ulcer (Vol I, page 70), in typhoid fever, and in various other infections. The hemorrhagic lesions in the pancreas would then be produced by ulcerations commencing in the endothelial lining of the vascular channels. These theories of pathogenesis have not been verified as yet by pathological examinations; indeed the hemorrhages and inflammatory changes are often so widespread that little pancreatic tissue remains for histological study.

It is important to observe that fat necrosis and hemorrhage in cases of pancreatitis go hand in hand; the latter is said by Dieulafoy never to exist without the former, though fat necrosis has been observed in numerous cases without any evidence of pancreatic hemorrhage. And as the area of fat necrosis may be widely disseminated, and not limited merely to the surface of the omentum, mesentery, etc., but situated deep within their substance; so, too, the hemorrhages of pancreatitis may be confined neither to the pancreas itself nor to a sanguineous peritoneal effusion, but often exist as distinct and separate hematomata, in the root of the mesentery, in the omentum, in the peripancreatic or perirenal fat, etc., etc. In other words both fat necrosis and hemorrhage may occur wherever the destructive pancreatic secretion is carried, whether its path is intraperitoneal, along the retroperitoneal tissues, through lymph-spaces, or through the blood stream; but this is theory, our knowledge at present not permitting of differentiation. Dieulafoy attempted to draw a sharp distinction between cases of hemorrhagic pancreatitis and cases of pancreatico-peritoneal hemorrhage, the former being infectious in origin and the latter merely toxic, caused by the extravasation of pancreatic juice which produces both hemorrhages and fat necrosis in various parts of the abdominal cavity; it arises not infrequently in the course of a chronic pancreatitis. It may thus be understood that a "pancreatic

apoplexy" so called was believed to arise without preceding infection or inflammation; but such cases, which once were regarded as not uncommon, appear to be of rare occurrence at the present day, possibly because closer study shows them to conform to the general law that infection precedes the hemorrhage.

But even though it is acknowledged that infection is the original cause of most cases of acute pancreatitis, the pancreatic lesions are not produced by direct invasion of the gland by bacteria, but chiefly by the circulating toxins. Truhart found bacteria in the pancreas in only fourteen out of seventy-four cases of acute pancreatitis which he studied; and though it has been pointed out by subsequent investigators that his studies did not exclude the presence of anaërobic bacteria, it has not been shown that these have any definite influence in producing acute hemorrhagic pancreatitis, except through their circulating toxins.

Clinical Ætiology. *Sex.*—The disease is more frequent in men than in women; according to Böhm, large statistics show that about 65 per cent. of cases occur in men. Peiser cited 121 cases of hemorrhagic and gangrenous pancreatitis, of which seventy-nine were in males and forty-four in females; Körte reported forty-one cases of acute pancreatitis, thirty being males and only fourteen females.

Age.—Most patients are of middle or later life, though cases are not unknown in young adults. Böhm states that of eight patients with acute pancreatitis operated on by Garrè, five were from thirty to fifty-two years of age, two were over sixty years, and one was only twenty-four years of age. In young persons it is said usually to occur in hard drinkers, who have developed arteriosclerosis before the usual time of life. Brewitt and Körte have each of them operated successfully on a patient of sixteen years with acute hemorrhagic pancreatitis.

Obesity usually is considered an important predisposing cause: among eighty-three cases of acute pancreatitis, collected by Williams and Busch, obesity was distinctly noted in fifty, and in many others the amount of adipose tissue was not mentioned at all.

Previous digestive disturbances have almost always existed; they may consist merely of gastro-duodenitis or of more serious affections.

Cholelithiasis, however, is not so frequent an accompaniment of acute as it is of chronic pancreatitis. Kehr observed acute pancreatitis in only 1 per cent. of his operations for gall-stone disease. The statistics collected by Quénu and Duval showed that, of cases of pancreatitis associated with gall-stone disease, 60 per cent. were cases of chronic pancreatitis, and 40 per cent. of acute pancreatitis (23 per cent. were cases of gangrenous or suppurative pancreatitis, and only 17 per cent. were cases of hemorrhagic pancreatitis). These figures agree very closely with those reported by other observers: Nötzel reported nine cases of acute pancreatitis, three of which were associated with gall-stones; of the eighty-three cases of acute pancreatitis collected by Williams and Busch, thirty-three (40 per cent.) were associated with gall-stones; and Egdahl found that gall-stones were present in forty-four out of 105 cases of acute pancreatitis. Opie has collected forty-three cases in which gall-stones and acute pancreatitis were associated, in nine of which a calculus had lodged near the termination of the bile-duct and may have permitted retrojection of bile into the duct of Wirsung (page 265). It has been suggested by Williams and Busch that the passage of a gall-stone into the duodenum may so dilate the intestinal orifice of the common bile-duct as to facilitate regurgitation of duodenal contents into the pancreatic duct. Numerous reports indicate that acute pancreatitis occurs by no means infrequently in a gland already the seat of chronic inflammation.

Typhoid Fever.—Musser observed acute hemorrhagic pancreatitis as a fatal complication of typhoid fever.

Trauma.—It is generally acknowledged that trauma may be a cause of hemorrhagic pancreatitis, the hemorrhage causing a place of lessened resistance and predisposing the gland to infection; moreover, the structure of the pancreas is such that very insignificant trauma may result in serious intraperitoneal hemorrhages. Cases of acute pancreatitis following trauma have been recorded, according to Carnot, by Littlewood, Lloyd, Rolleston, Pitt and Jacobson; Opie refers to additional cases recorded by Selberg, Schmidt, and Hahn; Robson and Cammidge have had one such patient under their care; and a case has been recorded by Ransohoff.

Morbid Anatomy.—On opening the abdomen early in the

course of a case of acute pancreatitis there may be found no exudate, and nothing to indicate disease of the pancreas, not even scattered areas of fat necrosis. At a later stage, however, perhaps after the lapse of only a few hours, there usually is found a sero-purulent exudate, sometimes blood-stained or even grumous in character. In nearly all cases areas of fat necrosis are found in the omentum, mesentery or peri-pancreatic fat. This exudate should not be interpreted as a direct extension from the diseased pancreas in all cases; it is the evidence that the general peritoneal cavity, and especially the omentum, is reacting to the pancreatic infection just as it does to infections arising in the gall-bladder, the appendix, the Fallopian tubes, etc. In cases where the exudate is blood-stained, however, and especially where there is extensive fat necrosis, it is probable, as previously noted (page 281), that the proteolytic an dsteatolytic ferments of the pancreas have escaped from their normal habitat, by way of the lymphatic spaces or possibly transperitoneally. The lesser as well as the greater peritoneal cavity usually is invaded by the exudate, which early in the course of pancreatitis is sero-purulent; later it becomes bloody, grumous, even chocolate colored; rarely it is frankly purulent. Hæmatomata may be observed in the root of the mesentery, around the pancreas, and in the peri-renal fat. The pancreas itself, in the very early stages of the disease, may present no very noteworthy macroscpoic changes, except that it may be enlarged; but very soon it becomes infiltrated with blood, which is conspicuous because in isolated spots separated by yellowish-white areas of normal pancreatic tissue. In the course of a few days, if death does not occur sooner, the pancreas may be converted into a reddish-black mass of necrotic fat and blood clots. The disorganization of the gland usually is so extensive that little of value can be learned from a microscopical study. The hemorrhages are interstitial, rarely invading the ducts.

If the patient lives, the lesions of gangrenous pancreatitis may be observed after the lapse of ten days or two weeks. In this stage a large portion of the pancreas, usually the body or tail, may be found almost completely detached from the surrounding tissues, lying as a slough in the retroperitoneal fat. The evidences of general peritoneal infection are now very slight, but

the entire lesser peritoneal cavity may be converted into an abscess containing foul-smelling, purulent, chocolate-colored exudate, with pieces of necrotic pancreas floating around loose in the fluid. In rare instances the stomach, jejunum or transverse colon may be perforated. In a case recorded by Chiari most of the pancreas (identified by Rokitansky) was passed from the rectum as a slough; this was also the case in a patient of Trafeyer. In a patient under the care of the senior author nearly the entire pancreas was discharged as a slough through a lumbar incision made for drainage. This case has been reported in full by Jurist.

Lesions of the biliary tract, a common accompaniment of acute pancreatitis, may also be observed on inspection of the abdomen at operation or *post-mortem*. They require no particular description in this place, but it is perhaps well to note that fat necrosis has been observed in a few cases apparently as the result of leakage of the pancreatic juice through a perforation or rupture of the biliary apparatus or duodenum (Richter), the pancreas itself not being diseased.

Symptoms.—Prodromal symptoms, in the nature of digestive disturbances, stomach or gall-stone cramps, etc., are said to exist in 70 per cent. of cases (Stich); but usually the acute symptoms arise so suddenly, and are of such an overwhelming nature, that the patient can give no detailed history of his previous condition, and such prodromal symptoms are discovered only by inquiries from the patient after recovery from operation, or from his friends after his death. The disease usually runs its course in from five to eight days, death occurring within a week in the great majority of cases without operation. In a few cases, however, if no operation is done, the symptoms abate, and when about the tenth day the hemorrhagic or purulent effusion has become localized, physical examination may enable a diagnosis of suppurative pancreatitis to be made (page 307).

The attack is characterized by both *abdominal* and *constitutional* symptoms. Of the first, *pain* and *vomiting* are the most important, and of the latter, *collapse*.

Pain.—This occurs suddenly in the epigastric region, and may be so severe as to cause faintness or collapse. It is a neuralgic pain at first, possibly as suggested by Guinard from pressure on

the solar plexus; sudden death may thus arise by inhibition of the heart. A rapid, but not sudden, death is more apt to be caused by toxæmia. The pain resembles in its colicky nature that due to intestinal obstruction, but it is extremely severe at its first onset, whereas the pain of obstruction frequently begins with mere twinges and becomes severe only after the lapse of hours. The pain of pancreatitis does not shift its position, but remains constantly epigastric, usually more to the left than the right of the median line. Pain may also be felt in the dorsal region, usually to the left of the spine, or in the left shoulder blade. It seems not unlikely in those cases attended by extremely severe pain, which cannot be relieved by morphine and which impels the patient to rise from the bed and walk around the room and frequently change his position, and which are not attended by marked collapse—that in these cases the hemorrhagic exudate is still confined by the capsule of the pancreas, not having broken through into the general peritoneal cavity; and that it is the latter event which brings on collapse. (Compare Elliot's second case.)

Vomiting is an early and important symptom. It follows closely after the initial pain and is repeated so frequently as to resemble that due to intestinal obstruction; but in the latter condition the vomiting is projectile, there is little or no nausea and retching, and the vomitus soon becomes bile-stained and then fæcal. In acute pancreatitis, on the other hand, the vomitus is never fæcal, and frequently (when the bile-duct is obstructed) it is not even bile-stained; there usually is considerable nausea and retching, the gastric and duodenal contents being brought up only with difficulty. *Hiccough* is a frequent symptom, is often repeated, and very persistent.

Jaundice is of rather frequent occurrence in acute pancreatitis, either from primary calculous obstruction, or secondarily from compression of the bile-duct by the diseased pancreas.

Emaciation.—This, which is extremely rapid, is very characteristic of pancreatitis in all its forms.

Collapse.—The extremities are cold, the face and hands are covered with cold sweat, the nose looks pinched; there is mental hebetude; sometimes great restlessness and thirst; delirium is rare, except in later stages. The symptoms of collapse probably are due to the absorption of toxins of the broken-down pancreatic

tissue (Egdahl); mechanical irritation of the peritoneum and stimulation of the cœliac plexus are also secondary causes.

Pulse.—In the presence of collapse this is rapid, feeble, and often indicates the hemorrhagic nature of the abdominal disease; but in a few cases where the excruciating pain has dominated the picture and collapse has been absent, the pulse has been slower than normal and quite strong (Elliot).

Temperature.—This may be subnormal and is seldom high, early in the disease. When the collapse passes off, and under the influence of the peritonitis, it may be high, and in cases of abscess of the pancreas may assume a hectic type.

Dyspnœa.—This, which perhaps is more accurately described as hypernœa, has been noted by several keen observers as a symptom characteristic of acute pancreatitis. It is probably due in part to mechanical interference by the engorged pancreas with the action of the diaphragm; in part to the severe pain, and in part to the toxæmia, as in the parallel case of uræmic dyspnœa.

Convulsions, followed by *coma* and *death*, were the chief symptoms noted in a case reported by Tomaschny: as the patient (an old woman with senile dementia) had never had epilepsy and as the kidneys were normal and there was no diabetes, Tomaschny concluded that the convulsions were either reflex, from pressure on the solar plexus, or toxæmic in origin; on the under surface of the dura mater there were some small yellow spots, perhaps areas of fat necrosis; this was also present in the peripancreatic fat.

Physical Signs.—Thorough physical examination usually is impossible at the first onset of the disease, owing to the extreme degree of abdominal pain and tenderness. *Inspection* shows a slightly distended epigastrium, with thoracic breathing; cyanosis is frequent and livid splotches on the surface of the abdomen have been noted several times. On *palpation*, the muscular rigidity is not found to be very marked; in a few cases gentle but persistent examination of the epigastrium with the warm flat hand has enabled the surgeon to detect that the swelling was diffuse, and not simply dependent on a distended stomach or colon (Robson and Cammidge). Gentle palpation in the left costo-iliac space may discover an abnormal fullness, as well as exquisite tenderness, from the pancreatic extravasation in the retro-peritoneal tissues.

Percussion may demonstrate an area of fullness in the region of the pancreas, between the ensiform process and the umbilicus.

Clinical Course.—After the extremely sudden onset of an attack of *acute pancreatitis*, characterized by violent epigastric pain, repeated vomiting, and collapse; the symptoms of peritonitis supervene, and are the dominant feature of the case during the second and third days of the disease. After this time, unless death occurs, the symptoms generally grow less severe, and the physical signs of *subacute pancreatitis* (gangrenous or suppurative stage) arise. The patient continues to be gravely ill, though suffering less intensely than at first; the stomach is unretentive, though vomiting usually is absent if entire abstinence from mouth feeding is persisted in, as it should be; emaciation is rapid; slight jaundice frequently is present; the pulse is weak and running; the temperature elevated (100° to 102° F.) and sometimes assumes a hectic type. In the epigastrium an indistinctly outlined and deep-lying tumor usually can be detected by palpation, and the rest of the abdomen may be no longer painful. The patient will now die of exhaustion, sepsis or secondary peritonitis from rupture of the pancreatic abscess, unless promptly relieved by operation.

Differential Diagnosis.—Unless a surgeon has seen previously two or three cases of acute pancreatitis, or unless he keeps the condition constantly in mind, it is seldom that a correct diagnosis is made before opening the abdomen; and it is generally admitted that any attempt to distinguish between the various forms of acute pancreatitis (hemorrhagic, suppurative, gangrenous), in the present state of our knowledge, is utterly futile.

The conditions for which acute pancreatitis is most often mistaken are biliary colic; acute intestinal obstruction; perforation of the stomach, duodenum or gall-bladder; and appendicitis.

Biliary colic presents many of the symptoms of acute pancreatitis, and a distinction may be very difficult. In acute pancreatitis, however, there usually is not a history of recurrent attacks; it is more apt to follow overeating than is biliary colic; and the pain though subject to exacerbations as in biliary colic; scarcely ever is entirely absent between these exacerbations, pain, moreover, is very much more intense in the pancreatic affection—indeed it is seldom relieved by the morphine which may be given in large and repeated doses under the impression that the

condition really is biliary colic. In pancreatitis there is collapse, cyanosis, and a sense of impending death, which seldom are noted in cases of biliary colic.

Intestinal Obstruction.—The difference in the character of the onset and in the nature of the pain have been mentioned already (page 295): in addition, there frequently is a history, in cases of acute intestinal obstruction, of a previous attack of peritonitis which might have left behind crippling adhesions, etc. The collapse is not so great as in pancreatitis, the temperature is not elevated, and the onset of peritonitis, with fever, wiry pulse, and distended abdomen is more delayed. The vomiting is projectile, with little or no nausea or retching, and rapidly becomes fæcal in the case of obstruction; whereas in pancreatitis the nausea is marked, the gastric and duodenal contents are brought up only with effort, and though the vomiting may be often repeated (every ten to fifteen minutes), fæcal vomiting scarcely ever is observed. A slight icteric tinge of the sclera is present sometimes in pancreatitis, but is very rare in intestinal obstruction; persistent absence of bile from the vomitus speaks in favor of pancreatitis. In both affections there nearly always is absolute constipation, and in both an evacuation of the lower bowel sometimes may be secured by the use of enemata even after the abdominal symptoms are well advanced. In most cases of pancreatitis, however, the *peritoneal* overshadow the *intestinal* symptoms, while the reverse is the case in the early hours of obstruction. Active peristalsis will be heard in obstruction, but will be absent in pancreatitis. Physical examination is more apt to be negative in cases of pancreatitis than in cases of intestinal obstruction, since here the existence of a tumor (intussusception, volvulus) sometimes may be demonstrated. Dyspnœa and cyanosis, if present, point to pancreatic disease. In addition to all the above differential points, it must be remembered that intestinal obstruction is more common in the young (intussusception, Meckel's diverticulum, appendicitis) and in the old (carcinoma, strangulated hernia, volvulus), while pancreatitis is most frequent in males of later middle life.

Perforation of the Stomach, Duodenum or Gall-bladder.—In these conditions it is possible in the vast majority of cases to obtain a history of gastric or biliary symptoms extending over a

number of years. In cases of perforation of the gall-bladder the patient almost invariably will have been confined to his bed for some days at least with upper abdominal symptoms, and frequently the distended gall-bladder can be recognized before perforation occurs. The onset of the attack in cases of perforation of a hollow viscus, though often quite as sudden as in acute pancreatitis, and though attended by very severe pain, is yet unattended by the marked collapse which is characteristic of the latter condition, and the vomiting is seldom repeated. As operation is demanded even at an earlier period in cases of perforation than in acute pancreatitis the differential diagnosis is of more academic than practical importance. (See also Vol. I, page 93).

Appendicitis.—While the initial pain in appendicitis is umbilical and colicky, as is often the case in acute pancreatitis; yet it is not so severe, collapse is rare, and vomiting is not repeated. The pain of appendicitis localizes itself in a few hours to the right iliac region, and any mass which forms will be here or in the pelvis; while a palpable mass forming as the acute symptoms of pancreatitis subside will be in the epigastric region, slightly to the left of the median line, or in the left loin. The age of the patients is different, and a history of previous attacks of appendicitis may throw further light on the diagnosis.

Gynæcological affections, such as ovarian tumors with twisted pedicle, rupture of a tubal pregnancy, etc., usually may be excluded by attention to the history and a careful pelvic examination.

Poisoning.—Leriche and Arnaud suggest that the cases of hyperacute pancreatitis ("pancreatic apoplexy"), followed by death in a few hours in a state of collapse, might be mistaken for poisoning by drugs and they recommend that pancreatitis be remembered as a cause of sudden death in cases having a medico-legal aspect.

Prognosis.—Acute pancreatitis is now regarded as a surgical disease; this is conceded by most physicians, and there are good reasons why it should be so. Under purely medical treatment the vast majority of patients with acute pancreatitis died. In 1908 Dreesmann collected case records from the past ten years, which showed that of thirty-six cases of acute pancreatitis treated without operation, only four recovered; a mortality of 88 per cent.; while of 118 cases subjected to operation fifty-three recovered, a mortal-

ity of only 55 per cent. The statistics collected by Ebner may be added: of twenty patients treated without operation, eighteen died, a mortality of 90 per cent.; of thirty-six patients subjected to operation, nineteen died, a mortality of 47.2 per cent.; and while, as Dreesmann suggests, such statistics as these no doubt give an unduly favorable record for the operative cases, since successful are more apt to be reported than unsuccessful operations, yet the difference between the medical and surgical results is so extreme that little further argument is needed.

With this limitation in mind, it is interesting to see the gradual improvement in operative results which is evidenced in the following statistics.

In 1906 Lenormant and Lecène found six recoveries among thirty-six operations (mortality 83.3 per cent.).

In 1909, Leriche and Arnaud found (since 1906) thirteen recoveries among thirty-nine operations (mortality 66.6 per cent.).

In 1911, Körte found among 118 isolated operations reported since 1905, seventy-three recoveries and forty-five deaths (mortality 38 per cent.).

It has been argued that patients who are finally subjected to operation sometimes give a history of having passed through similar attacks before, recovering without operation; but it is even more difficult to diagnose an attack of acute pancreatitis from vague descriptions given by the patient of a previous illness than it is when we see him during the attack; and it seems not impossible that some of these acute attacks from which the patients are alleged to have recovered have been attacks of biliary disease. Yet it is true that some of the operations followed by recovery (Halsted, Dick) consisted in little more than opening the belly and sewing it up again; but one or two cases are not enough to controvert the dictum that without operation the prognosis is shockingly bad.

The first recovery after operation with drainage is credited to Hahn (1900).

There is little doubt that as experience accumulates it will be possible to recognize milder attacks of acute pancreatitis than the fulminating cases which hitherto have occupied the attention of surgeons.

As to the time at which operation shall be undertaken, surgeons are gradually becoming unanimous in their view that immediate intervention offers most chances of success. Bunge, Böhm, Ebner, Elösser, Kehr, Mayo, Moynihan, Nötzell, Robson, and others whose experience has been greatest in the treatment of acute pancreatitis, all urge immediate operation; and even Körte, who at first (1898) advised waiting until the subacute stage on the ground that no operation could be of benefit in patients so acutely ill, revised his teaching in 1907, and now asserts his opinion that operation as soon as possible after the onset of symptoms affords the best chance of cure. And as we learn more of the pathology of the affection, this seems the only reasonable ground to take: the conditions in acute pancreatitis are in many respects similar to those present in traumatic lesions of this region—escape of pancreatic juice into the peri-pancreatic tissues and the general peritoneal cavity, and hemorrhage. If the case were traumatic in origin, no one would counsel delay in opening the abdomen and attempting to evacuate the toxic fluid (absorption of which causes the collapse, etc.) and to check the bleeding by tampon or suture. As a matter of fact, however, very few operations have been done within a few hours of the onset of the attack; usually the patient does not come under surgical care until the second or third day of the disease,[1] and we believe that under such circumstances, as in other cases of diffuse peritonitis, it is safer sometimes to encourage localization of the process before instituting drainage. But it should be distinctly understood that by urging postponement of operation at this stage we do not mean to leave the patient alone until he is nearly moribund from sepsis: sometimes it may be possible to wait until a well-localized tumefaction indicates the presence of an abscess or of gangrene; but whenever a case is seen before symptoms of diffuse peritonitis arise, the surgeon should lose no time in opening the abdomen to evacuate the toxic extravasation. In general, we believe that immediate operation should be done, unless such a course manifestly would hasten death; under such circumstances, and when the patient is first seen when diffuse peritonitis is well advanced, it will be safer to postpone operation, rely-

[1] Thus in Hahn's case, already alluded to, operation was not undertaken until three days after the onset of the disease; Bunge's patient, quoted far and wide as an incontrovertible proof of the value of immediate operation, was not operated on until sixty hours after the onset of the attack.

ing on the "Ochsner treatment" to encourage localization of the process.

It is almost impossible to secure figures which will give any clear indication of the truth in this matter, for the case reports as a rule do not give the number of hours elapsed between onset of the attack and operation, nor do they clearly define the stage of disease (hemorrhagic or suppurative) in which operation is undertaken; and even if such figures were compiled, from scattered case reports, they could not convey the real truth, because successful operations are always reported sooner than unsuccessful. Nor would the patients who had died without operation be included in such statistics. Körte has recently attempted to show the results obtained by those who have reported more than one operation, (presumably their entire experience in each instance); he collected in this manner reports of 103 operations: among these, forty-one patients recovered, and sixty-two died, a mortality of 56.9 per cent. Körte's own experience (embracing ten cases of his colleague Brentano) includes forty-four cases of acute pancreatitis, in the period from 1890 to 1910. Only cases where the diagnosis was proved at operation or at autopsy are counted. In six cases no operation was done, and all the patients died. In thirty-eight cases operation was done; in four cases the gall-bladder was drained and nothing was done to the pancreas; all the patients died. In thirty-four cases the usual operation on the pancreas was done, with eighteen recoveries and sixteen deaths (47 per cent. mortality).

KÖRTE'S OPERATIONS.

	Total.	Recovered.	Died.	Mortality. Per Cent.
In the first week of the disease	12	8	4	33.3
In the second week of the disease	4	3	1	25.0
In the third week of the disease	7	4	3	43.0
In the fourth week of the disease	7	3	4	57.0
From the fifth to the seventh week	4	0	4	100.0
	34	18	16	47.0

There are few, if any, surgeons who have had so extensive an experience with this disease as has Körte. In the annexed table we have assembled the statistics of those surgeons whose

experience most nearly approaches his. The senior author reported in 1909 the histories of six patients who had been under his care up to that time. In all operation, not always immediate, was done, with three recoveries and three deaths. Since that time he has encountered 13 more cases.

STATISTICS OF ACUTE PANCREATITIS.

Author.	Total Number of Cases.				No Operation.				Operation.			
	Total.	Recovered.	Died.	Mort. %	Total.	Recovered.	Died.	Mort. %	Total.	Recovered.	Died.	Mort. %
Balch and Smith (1910)....	21	5	16	76	6	0	6	100	15	5	10	66
Bode (1911).....	5	1	4	80	..	..	..	.....	5	1	4	80
Böhm (1904) Ehrich (1898) (Garrè).	8	1	7	87	4	0	4	100	4	1	3	75
Borelius (1911).....	11	4	7	64	3	0	3	100	8	4	4	50
Bornhaupt (1907).....	4	1	3	75	1	0	1	100	3	1	2	66
Deaver (1912).....	19	11	8	42	8	6	2	25[1]	11	5	6	54
Dreesmann (1911).....	14	7	7	50	3	2	1	33	11	6	5	45
Elösser (Czerny) (1908)....	7	3	4	57	..	..	..	.....	7	3	4	57
Frank and Price (1912)....	9	1	8	89	..	..	..	.....	9	1	8	89
Keefe (1910).....	8	2	6	75	1	0	1	100	7	2	5	71
Körte and Brentano (1911)	44	18	26	59	6	0	6	100	38	18	20	53
Mettin (Neumann) (1912)..	22	6	16	73	..	..	..	.....	22	6	16	73
Moynihan (1911).....	11	7	4	36	..	..	..	.....	11	7	4	36
Robson (1911).....	10	7	3	30	..	..	..	.....	10	7	3	30

Treatment.—When operation is undertaken, a certain definite technique should be followed. In cases where the diagnosis is uncertain, the surgeon may make his first incision in the hypogastric region; but if he is an acute clinician it usually will be possible for him to determine before beginning whether the seat of disease is in the lower or upper abdomen. In any case, however, pancreatic disease is to be suspected so soon as the abdomen is opened if there is a bloody turbid exudate, or if there is fat necrosis; marked distention of the transverse colon, without apparent obstruction, is another valuable sign. If only an exploratory hypogastric incision has been made (as is best in cases of uncertain diagnosis), it should be temporarily plugged

[1] The diagnosis in patients who recover without operation often is uncertain.

with gauze; an incision is then made in the epigastrium, through the left or right rectus. The pancreatic lesion may now be self-evident; and the stomach may be displaced by the swollen gland simply forward, or upward or downward; rarely the colon will be displaced upward, and the pancreas prove most accessible through the transverse meso-colon. When a choice is possible, the surgeon should aim to expose the pancreas through the gastro-colic omentum, dividing this structure between the gastro-epiploic vessels and the colon, as described in Vol. I, page 323 (Fig. 21); but if the pancreas bulges through the gastro-hepatic omentum or through the transverse mesocolon, it should be approached by that route. Bunge recommended in all cases that an additional opening should be made for drainage through the meso-colon; but usually this is not advisable.

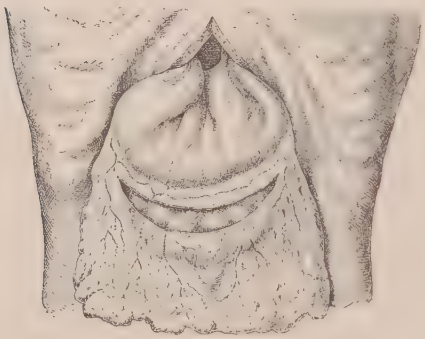

Fig. 21.—Wide Opening of the Gastrocolic Omentum to Explore the Pancreas.

Having thus exposed the pancreas itself, many surgeons have been content to conclude their intervention at this point by tamponing the lesser peritoneal cavity, without opening the capsule of the pancreas. Stieda, Nötzel and others who recommend this plan contend that incision of the swollen hemorrhagic pancreas may lead to uncontrollable hemorrhage, and will give exit to exceedingly toxic substances which will be more rapidly absorbed by the peritoneum, and thus more quickly destroy the patient, than if they were still confined to the substance of the inflamed gland.

Direct incision of the pancreas was first proposed in 1893 by Nimier, who suggested controlling the hemorrhage by the actual cautery or by gauze packing. It appears to have been put into execution first by C. B. Porter in February, 1903, closely followed (December, 1903) by Muspratt, both patients recovering.

As far as our own experience goes, and from what can be gathered from case reports of other surgeons, there is no object

in incising the pancreas unless it appears distended, or unless there is a hæmatoma present. In several cases operated on in the hemorrhagic stage (Bunge, Hahn, Coenen, Nötzel) the evidences of intra-pancreatic damage have been comparatively slight, and recovery has followed mere tamponade of the lesser peritoneal cavity. In Coenen's patient (Case 1) there was no exudate at the time of operation, but seven days after operation there occurred a profuse discharge of pancreatic secretion from the wound, with sloughs of the pancreas; and in Nötzel's patient (Case 3) no cause for the peritonitis was found at the time of operation, so the wound was tamponed, but reopened during the subacute stage when the pancreatic exudate was evacuated. The cases recorded by Bunge and Hahn are similar.

On the other hand we do not think that such great fear of systemic poisoning need be felt if the distended pancreatic capsule is incised to give exit to dammed-up secretions or blood clots; indeed it seems not improbable that there is less danger of absorption from the serous surface of the peritoneum than from the retroperitoneal cellular tissues. In every case, of course, the pancreas should be opened only after carefully protecting the general peritoneal cavity by a coffer-dam of gauze; and if there is any localized collection of fluid, this may well be evacuated first by an aspirator, still further to decrease the chance of soiling the peritoneum, the abscess cavity or hæmatoma being formally incised only after its contents have been withdrawn.

Bircher successfully excised the entire tail of the pancreas which was the seat of a hæmatoma on the third day after onset of symptoms; and he urges excision whenever possible, as the removal of the focus of disease, as of a gangrenous appendix, should accelerate the cure.

Another question in dispute is whether or not any treatment of biliary complications should be undertaken when the abdomen is opened in a case of acute pancreatitis. This was urged as a measure of routine by Ebner, but the proper course, it seems to us, depends upon the state of the biliary tract and the condition of the patient. Should the latter warrant such interference, we see no good reason for postponing drainage of the gall-bladder if this is easily accessible and is the seat of acute cholecystitis, or

contains calculi; if on the other hand the gall-bladder is buried in adhesions, if there is doubt as to its being acutely diseased, or if for any reason the calculi present are particularly inaccessible, it no doubt would be proper to postpone to a more opportune time any indicated operative treatment of the biliary tract, even if the condition of the patient were reasonably good. But the surgeon should not forget that a diseased biliary tract will require treatment sooner or later.

Many surgeons still urge free irrigation of the peritoneal cavity with saline solution; they claim that not only is it important to secure evacuation of all the exudate, but that owing to its toxic nature it is extremely desirable to dilute it. As cultures from the bloody exudate have in a large number of cases shown it to be sterile, and as sufficient evacuation is secured through absorption by the gauze packs used during the operation, it seems to us quite unnecessary to irrigate the abdominal cavity; the practice probably is not as undesirable as in cases of appendicular peritonitis, but that is the most that can be said in its favor.

We may sum up the technique of operation for acute pancreatitis as follows: through an epigastric incision isolate the pancreatic region by gauze packs; if a collection of fluid exists, evacuate it by aspiration; expose the pancreas preferably through the gastro-colic omentum, and if it presents no gross lesions do not incise it, but merely tampon the lesser peritoneal cavity; if there is a hæmatoma or abscess in the pancreas, incise its capsule, and with a blunt instrument carry the incision into the substance of the gland, to secure drainage of all pockets of pus, etc. Then tampon the incision into the pancreas, using a large rubber tube for drainage in the center of the tampons. In some cases a counter-incision in the left loin will be desirable, as in cases of subacute pancreatitis (page 399). Should a hypogastric incision have been made at first through inadvertence, it should be plugged temporarily with gauze, and may be used at the conclusion of the operation for pelvic drainage, if this seems indicated; but in most instances it is better to close it entirely. The biliary tract should not be interfered with except for very positive indications and only when the patient is in reasonably good condition.

After-treatment does not differ materially from that employed in other acute lesions of the upper abdomen. If the patient

does well, it may be expected that there will be rather free discharge from the drains, and that after four or five days some evidence of pancreatic fluid will be found on the dressings. Should meteorism, vomiting, fever, etc., reappear some days after operation, the patient having done well meantime, the surgeon should suspect either a fresh pancreatic hemorrhage or at least that his drains are blocking rather than aiding the evacuation of sloughs or pus from the pancreas; in such circumstances a new operation is indicated, to establish better drainage, as recommended by Leriche and Arnaud (loc. cit., p. 834); but this should not be undertaken until lavage of the stomach and large bowel have proved ineffectual in arresting the symptoms.

After the first few days the case will resemble one of subacute pancreatitis, so that a more detailed account of the after-treatment may well be postponed until that subject has been considered.

Subacute Pancreatitis: Abscess and Gangrene of the Pancreas.

Abscess or gangrene of the pancreas usually arises as a sequel of acute inflammation of the gland; very seldom is abscess a primary condition, and almost never does gangrene occur except during the subacute stage which succeeds to an attack of "acute hemorrhagic pancreatitis." Some writers attempt to distinguish between *suppurative pancreatitis* and *abscess of the pancreas*. Robson, for instance, compares the former to acute suppurative mastitis and the latter to mammary abscess; but though such a distinction is quite proper theoretically we doubt the ability of surgeons to make a clinical distinction with the means at present at our command; and it is, we believe, also impossible to distinguish between the *suppurative* and *gangrenous* forms of the disease.

The **diagnosis,** in fact, rests more upon the *clinical course* (page 297) of the affection, and upon the *physical signs*, than upon the symptoms which the patient presents. The evidences of deep-seated swelling in the epigastric region, following upon an acute peritoneal attack, always should make the surgeon suspicious of pancreatic trouble; but the physical signs often are very like those of *subphrenic abscess* (Vol. 1, page 443), which condition indeed may be due to pancreatic disease. Among the forty-four cases of sup-

puration in the lesser peritoneal sac studied in 1904 by Michel and Gross twenty-four were certainly, and four probably, caused by pancreatic disease, six by affections of stomach and œsophagus, one by perforation of the colon, seven by splenic affections; while in two the cause was undetermined.

Pancreatic abscesses may reach a large size: Fasano reported one containing four liters of pus, and Coenen's fifth patient had one which contained more than a liter and a half. These abscesses have a tendency to point (1) in the lumbar regions (Brentano and Rotter have reported cases pointing simultaneously in both loins); (2) anteriorly in the abdominal region; or (3) in the left thoracic region (Guinard). In the first instance the signs of *perinephric abscess* will be simulated, and in the third, those of *pyopneumothorax* or *subphrenic abscess;* while those pointing anteriorly may be mistaken for subphrenic abscess due to subacute perforation of the stomach. In all cases, however, there usually will be the history of sudden hyperacute onset, with collapse, peritoneal symptoms, etc., and then as the patient gradually recovers from the first violence of the attack there will be the development of these secondary signs of upper abdominal or subphrenic suppuration; and it is on the clinical history of the disease, *plus* these physical signs, and not on one or the other alone, that a diagnosis must be based.

Treatment consists in evacuation of the abscess and drainage of the pancreas, with removal of such sloughs as are already detached. The abscess should be approached where it is about to point; but if the surgeon wishes to save his patient's life he must not wait for subcutaneous fluctuation to enable him to determine this point. In many cases it will be best to make an exploratory epigastric incision, so soon as it is determined that some form of subacute pancreatitis is present, and then to make a counter-incision in the loin or elsewhere, as may be shown by this exploration to be best.

1. *The Abdominal Route.*—After opening the abdomen through the left rectus, in the epigastric region, the intestines are walled off with gauze packs, and the pancreas is palpated; if at all feasible, the lumbar route is now employed for drainage. If this does not seem possible, access to the pancreas is gained preferably through the gastro-colic omentum. If a frank abscess is found, it should be evacuated by aspiration, and when emptied, the abscess wall

should be incised. Sloughs which are entirely loose are then extracted, but those which are still attached even in part should not be detached roughly for fear of exciting hemorrhage or spreading infection. The abscess cavity is then tamponed, a rubber drainage tube being placed in the midst of the gauze packs. The gall-bladder should then be inspected, and if acutely inflamed or containing calculi, should be drained, unless distinct contraindications exist. Such drainage is better done through a stab wound on the right. The gauze used as coffer-dam to protect the general peritoneal cavity during the operation, is then removed and the abdominal incision is closed not too tightly around the gauze and tube drainage. It is not necessary nor is it advisable to attempt to suture the walls of the abscess cavity or the edges of the opening in the gastro-colic omentum to the parietal peritoneum. Brewer reports two successful operations by the abdominal route, and it is preferred by Désjardins in all cases; but Brentano, who has operated on six patients with pancreatic necrosis, with only two deaths, employed the abdominal route in only one case, which terminated fatally.

2. *The Lumbar Route.*—This we believe to be preferable to the abdominal route whenever it can be employed. As mentioned above the applicability of this route sometimes cannot be determined except by exploratory laparotomy. But if bulging, or even marked tenderness without other signs, can be detected in the left costovertebral angle, an incision as for kidney operations should be made here without previously opening the abdomen. Care is necessary not to penetrate the peritoneum; the dissection is carried beneath the lower pole of the kidney, and by burrowing with the finger toward the middle line, little difficulty should be experienced in locating the pancreatic exudate. The abscess is freely opened, and drained with tube and gauze. As already mentioned Brentano and Rotter have each opened a pancreatic abscess through both loins simultaneously. The lumbar route has been employed several times successfully by the senior author (p. 303).

3. *The Thoracic Route.*—This has been particularly commended by Guinard, by whom it has been employed twice with success. In the first case (1898) he operated two months and a half after the onset of acute pancreatitis on a patient who had already had a dis-

charge of pus through the vagina, with sudden subsidence of the pancreatic tumor; when this refilled operation was undertaken and the abscess was reached by the usual transpleural route employed for subphrenic and hepatic abscess. (See Hepatic Abscess, page 181.) In his second case (1907) operation was done two weeks after the acute onset, for signs resembling left pyopneumothorax, or subphrenic abscess. Körte has used the transpleural route in three cases, one patient recovering.

Prognosis.—If the condition is recognized and operation is done at the appropriate time, the prognosis is not bad. But the surgeon must be constantly on the alert, so as not to let the opportune time for surgical intervention slip past. As soon as the acute process shows signs of localization, but before hectic temperature, chills, sweats, emaciation, etc., show that septic absorption is going on, the pancreas should be drained. Though cases have been reported in which spontaneous discharge of pancreatic abscesses has occurred (through the stomach, rectum, vagina, and in the lumbar and iliac regions), and though in one or two of these cases the patients have recovered without operation, no such termination should be waited for; and though even sloughs of nearly the entire pancreas have been discharged through the rectum (Trafeyer, Chiari), it is not too much to say that were such an event to occur to-day the patient would recover more by good luck than by good management.

That the mortality after operation in the subacute stage, as reported by many surgeons, is lower than that attending early operation, is not an argument in favor of delay in resorting to operation. It indicates merely that the patients who have survived through the earlier stages of the disease have been less seriously ill from the first or have possessed better recuperative powers. Robson gives the mortality for acute pancreatitis as 61 per cent., and that for the subacute stages as 36 per cent. Villar gives 78 per cent. as the mortality for hemorrhagic pancreatitis, 38 per cent. for the suppurative, and 49 per cent. for the gangrenous form. Mettin reports a mortality of 71 per cent., for operations done during the hemorrhagic or suppurative stages; and of 66 per cent. for those done during the stage of necrosis.

After-treatment.—This comprises both *local* and *general* treat-

ment. The outer dressings will require frequent changing at first, but the gauze drainage should not be removed until it becomes loose of itself; when this stage is reached, there is no object, so long as free drainage exists, in keeping the wound widely open. Sloughs may be discharged from time to time, and the surgeon usually finds that closure of the wound is less rapid than he had anticipated, and that his best efforts will have to be directed not at keeping it open but in encouraging it to heal. Constitutional treatment is important, the patients becoming rapidly emaciated and requiring stimulants and nutritious food. Antidiabetic diet should be used, consisting almost entirely of fats and albumen, as advised by Wohlgemuth, and sodium bicarbonate should be administered during meals to lessen the gastric acidity, which is an excitant of pancreatic secretion; or pankreon may be given to substitute the pancreatic secretion lost through the fistula. Erepton proved effective in closing a pancreatic fistula in a case of rupture of the pancreas reported by Kroiss; about 100 grammes were given daily, 20 grammes at a dose, by mouth in much sweetened coffee or warm milk; or by rectum in doses of 50 grammes. In this patient, no change occurred in the profuse discharge from the fistula for a week after the administration of erepton was begun; then the fistula closed rapidly in three days and remained healed.

REFERENCES.

Balch and Smith: Boston M. and S. J., 1910, clxiii, 384.

Bode: Beitr. z. klin. Chir., 1911, lxxi, 610.

Böhm: Beitr. z. klin. Chir., 1904, xliii, 694.

Bornhaupt: Arch. f. klin. Chir., 1907, lxxxii, 209; St. Petersb. med. Woch., 1909, xxxiv, 115.

Brentano: Zentr. f. Chir., 1909, xxxvi, 540.

Brewitt: Berl. klin. Woch., 1908, xlv, 531.

Borelius: Beitr. z. kl. Chir., 1911, lxxiii, 261.

Bircher: Arch. f. klin. Chir., 1910, xciii, 415.

Bunge: Verhandl. d. Deutsch. Gesellsch. f. Chir., 1903, xxxii, 315.

Brewer: Surg. Gyn. and Obst., 1907, v, 344.

Carnot: "Mal. du Pancréas," Paris, 1908, p. 185.

Chiari: Int. Med. Congr., Lisbon, 1906, cited by Williams and Busch, loc. cit.

Coenen: Berl. klin. Woch., 1910, ii, 2177.

Deaver: Amer. J. Med. Soc., 1909, cxxxviii, 829.

Desjardins: Thèse de Paris, 1905.

Dick: Edinb. Med. J., 1910, v, 217.

Dieulafoy: Pathol. Int., 15e. éd., Paris, 1908, ii, 1044.
Dreifuss: Deut. Zeit. f. Chir., 1908, xciii, 432.
Dreesmann: Med. Klinik, 1908, ii, 1458, 1496, 1531; Ibid., 1911, vii, 993.
Ebner: Volkmann's Samml. klin. Vorträge, 1907, Nos. 452 and 453. (Chir. Nos. 128 and 129).
Egdahl: Bull. Johns Hopkins Hosp., 1907, xviii, 130.
Egdahl: Jour. of Exper. Med., 1907, ix, 385.
Ehrich: Beitr. z. klin. Chir., 1898, xx, 315.
Elliot: West London Med. J., 1910, xv, 291.
Elösser: Mitth. a. d. Grenzgeb. d. Med. u. Chir., 1907–08, xviii, 195.
Fasano: Zentr. f. Chir., 1908, xxxv, 1378.
Fitz: Trans. Congr. Amer. Phys. and Surg., 1903, vi. 41.
Frank and Price: Personal communication.
Guinard: Bull. et. Mém. Soc. Chir., Paris, 1898, xxiv, 381.
Guinard: Bull. et. Mém. Soc. Chir., Paris, 1907, xxxiii, 197.
Hagen: Wurzb. Abhandl. a. d. Gesamtgeb. d. prakt. Med., 1909, ix, 285.
Hahn: Deutsch. Zeit. f. Chir., 1900, lviii, 1.
Hahn: Deutsch. med. Woch., 1901, xxvii, V–B. 5.
Halsted: Bull. Johns Hopkins Hosp., 1901, xii, 179. (Reference to successful case op. on 11 yrs. previously).
Halsted: see Körte: Verletzungen u. Erkrank d. Pancreas (Deutsche Chir. Lief. 45 d) Stuttgart, 1898, S. 171.
Jurist: Amer. J. Med. Soc., 1909, cxxxviii, 180.
Keefe: Amer. J. Obst., 1910, lxii, 1050.
Kehr: Mitth. a. d. Grenzgeb. d. Med. u. Chir., 1909, xx, 45.
Körte: Arch. f. klin. Chir., 1911, xcvi, 557; Annals of Surg., 1912, i, 23.
Kroiss: Beitr. z. klin. Chir., 1911, lxxvi, 477.
Lenornant and Lecène: Rev. de Gyn. et de Chir. Abd., 1906, x, 1072.
Leriche and Arnaud: Rev. de Gyn. et de Chir. Abd., 1909, xiii, 795.
Mettin: Deut. Zeit. f. Chir., 1912, cxv, 281.
Mikulicz: Mitth. a. d. Grenzgeb. d. Med. u. Chir., 1903, xii, 1.
Michel and Gross: Rev. de Gyn. et de Chir. Abd., 1904, viii, 45.
Monnier: Corr.–Bl. f. Schweizer Aerzte, 1911, xli, 225.
Muspratt: Brit. Med. J., 1904, i, 304.
Moynihan: Brit. Med. J., 1911, i, 735.
Musser: Trans. Ass. Am. Phys., 1908, xxiii, 555.
Notzel: Beitr. z. klin. Chir., 1908, lvii, 734.
Nimier: Rev. de Chir., 1893, xiii, 634.
Opie: Osler's Mod. Med., Phila., 1908, v, 635.
Porter: cited by Mikulicz, Trans. Congr. Amer. Phys. and Surg., 1903, vi, 76.
Poucher: Amer. J. Obst., 1910, lxii, 974.
Quénu et Duval: Rev. de Chir., 1905, xxxii, 401.
Ransohoff: Trans. South. Surg. and Gyn. Assoc., 1909, xxii, 112.
Richter: Quarterly Bull. Northwestern Univ. Med. School, Chicago, 1910[1].

[1] This work has not been accessible to us.

Robson and Cammidge: The Pancreas, its Surg. and Path., Phila, 1907.
Robson's operative statistics, cited from Körte, l. c. 1911.
Rotter: Zentr. f. Chir., 1909, xxxvi, 541.
Stich: cited by Böhm, loc. cit.
Stiedel: quoted by Gobiet, Wien. klin. Woch., 1910, xxiii, 1672.
Thomas: Brit. Med. J., 1907, i, 201.
Truhart: cited by Carnot, loc. cit.
Truhart: Wien. med. Woch., 1906, lvi, 2097.
Williams and Busch: Trans. Assoc. Amer. Phys., 1907, xxii, 310.
Villard and Stephani: Lyon Méd., 1909, cxiii, 1138.
Wohlgemuth and Noguchi: Berl. klin. Woch., 1912, xlix, 1069; also Abstract in Jour. A. M. A., 1912, ii, 122.

Chronic Pancreatitis.

Chronic pancreatitis is an inflammation of the pancreas resulting from bacterial infection not severe enough to produce the acute type of inflammation which has already been studied. The channels of infection in cases of pancreatitis have been considered at length at page 259.

It has been usual to classify chronic pancreatitis as catarrhal or interstitial, according as the ducts or the interstitial tissues are especially affected.

Chronic Catarrhal Pancreatitis.—In this form, which is termed also *sialodochitis pancreatica*, the infection is believed to reach the ducts of the pancreas by way of the duodenum and common bile-duct, for the reasons already set forth (page 263). This form of pancreatitis is considered by Mayo Robson and others to be of frequent occurrence. The number of cases available for microscopical study has been small, necessarily, as death seldom occurs at this stage of the disease, and even if it did attention might not be directed particularly to the pancreas. Some cases, however, have been studied; and while the usual lesions present in catarrhal inflammation of glandular organs have been found (cloudy swelling, desquamation, etc.) there also have been present interstitial changes, which in our belief, as will be stated presently, are of more import than the strictly catarrhal features of the inflammation.

The *symptoms* of chronic catarrhal pancreatitis, according to Mayo Robson, usually are not to be distinguished from those

which he describes as due to chronic interstitial pancreatitis; and the *treatment* is the same.

Pancreatic Lymphangeitis.—Reference to this condition at page 266 has indicated our belief that most cases classed together under the general term of "Chronic pancreatitis" are at first really cases of pancreatic lymphangeitis, the infection being propagated from the gall-bladder and bile-ducts or from the pyloric region of the intestine along their efferent lymph-channels, which come into intimate relation with those surrounding and imbedded in the head of the pancreas. Not only the grosser lesions of the gall-bladder and juxta-pyloric regions, such as cholecystitis and gastric or duodenal ulcer, should be considered in this connection, but also those more frequent minor infectious processes which pathologically are termed catarrhal and which usually fail of clinical recognition, except as "indigestion" or "dyspepsia." We are accustomed now to recognize extremely mild grades of inflammation of the gall-bladder by the presence of ever so slight thickening, diminution in lustre, increased opacity, or inspissation and tarry character of its contents. If the same attentive scrutiny were directed to the stomach and duodenum, slight thickening, local puckering or distortion and increased opacity in many cases otherwise obscure might reveal the present or past existence of inflammation. As evidence of previous inflammation we have observed dimpling of the wall, at times suggestive of the starting point of an intussusception. Filmy adhesions, so well described by R. T. Morris as "cob-webs in the attic of the abdomen," should not pass unnoticed; they are clear evidence of a previous inflammatory process. Less noticeable, but significant when observed, is a peculiar streaked, opaque appearance affecting a very limited portion of the gastro-duodenal wall and often fading away in adjacent peritoneal attachments. Of course the grosser cicatrices of healed ulcers are readily seen, but these less conspicuous changes should be looked for, since they indicate in many instances the previous existence of interstitial inflammatory processes. Catarrhal inflammation, fissures, and even early ulcers, may exist without external evidence of their presence; and yet they act as portals of bacterial invasion as proved by the enlargement of the regional lymph-nodes.

The lymphatics of the pancreas have been studied thoroughly

by Bartels and the following description is taken largely from his work, and from that of Franke, who has studied the relation of the biliary lymphatics to the pancreas. The pancreas, unlike certain other organs, possesses no great hilum through which pass the afferent and efferent blood- and lymph-vessels. These vessels are distributed to the pancreas in a *more or less segmental manner*. Thus the blood-vessels which supply the head of the pancreas are quite distinct in origin from those which supply the rest of the gland; and these latter, derived from the splenic artery, supply each its own more or less isolated segment, though of course intra-pancreatic anastomoses exist. In a like manner the pancreatic lymph-channels, arising in the interior of the gland, come to its surface by many different and quite distinct trunks. These trunks communicate with various groups of lymph-nodes around the pancreas, the most important of which are thus enumerated by Bartels: the classification is made according to the situation of the lymph-nodes and the portion of the pancreas which they drain.

Group I. The Pancreatico-splenic Group.—These lymph-nodes are situated above and behind the tail of the pancreas in the hilum of the spleen. They lie along the splenic vessels between the layers of the gastro-splenic omentum. The lymph-vessels coming out of the tail of the pancreas run to these nodes.

Group II. The Superior Pancreatic Group.—These lymph-nodes lie along the superior border of the pancreas and receive the lymph-vessels coming from the upper portion of the body of the pancreas. There is no sharp differentiation between this and the pancreatico-splenic group and their vessels anastomose freely. Included in the superior pancreatic group are the nodes lying behind the pylorus, often spoken of as the retropyloric nodes. In addition to these connections the lymph-vessels from the upper border of the pancreas have connections with the superior gastric nodes (including the cardiac) and also with the hepatic nodes.

Group III. The Inferior Pancreatic Group.—This consists of a small number of lymph-nodes situated along the lower border of the pancreas. From the lower portion of the body of the pancreas lymphatic vessels run to these nodes and also to the aortic, mesenteric and mesocolic groups.

Group IV. The Pancreatico-duodenal Group, Anterior and

Posterior.—These lymph-nodes lie around the head of the pancreas and receive vessels from the pancreas and from the duodenum.

In addition to indirect communication by way of the lymphatic nodes there is also direct communication by anastomosis between the duodenal and pancreatic lymphatic vessels. In addition to this direct communication there are also anastomoses between the pancreatic lymph-vessels and those from the duodenum running to the mesenteric nodes. Of especial interest from the clinical standpoint is the communication shown to exist between the vessels from the head of the pancreas and a lymph-node situated between the portal vein and the common duct. Lymphatics run from the liver to this node.

It is in this particular that Franke's work supplements that of Bartels. Franke showed by Gerota injections that the lymphatic vessels of the gall-bladder run to nodes which lie to the left of the head of the pancreas near the common duct. On the way the greater part of these vessels are in relation with a node, which, when present, is situated at the neck of the gall-bladder. By injection from the gall-bladder Franke filled a plexus of lymph-vessels situated on the posterior surface of the head of the pancreas.

These four groups of lymph-nodes receive tributaries not only from corresponding portions of the pancreas but also from neighboring viscera, the spleen, stomach, left adrenal, liver and duodenum. The efferent vessels from the pancreatic nodes run to the parietal lymph-node groups of the abdominal cavity.

Lymphatic-born infection from these areas in order to reach the pancreas must in most cases stem the efferent current from the pancreas and force the valves. Here, as elsewhere in the body, the lymphatics are wonderfully efficient in preventing this outcome. Only when the intercommunications of the pancreatic lymphatics with those of adjacent organs are most intimate, short in their course, and unprotected by intervening lymph-nodes, does peril arise. Such an extremely intimate relationship Bartels has shown to exist between the lymphatics of the head of the pancreas and the adjacent duodenum; and more recently Franke has demonstrated that the same is true of the lymphatics coming from the gall-bladder. Should thrombo-lymphangeitis occur in any of these vessels as the result of infection originating in the duodenum or gall-bladder, reversal of

the lymph-current may occur in the pancreatic lymph-channels, in an effort to establish a collateral circulation; and in this way infection may be propagated from the gall-bladder, bile-ducts, or duodenum to the pancreas. A sufficiently severe infection might not even wait for reversal of the lymphatic current, but might rapidly invade the pancreas and infect not only the lymphatic tissues but the surrounding structures as well. The intrinsic lymph-vessels of the pancreas run in the interlobular septa, and it is here that the effects of this type of inflammation should be most manifest; and in accordance with this explanation is the fact that it is the interlobular form of pancreatitis which is associated with inflammatory lesions of neighboring viscera, in contrast with the interacinar sclerosis, which, as pointed out by Opie, appears to bear no such relation to local inflammation. It is true of course that the interlobular distribution of the inflammatory changes might be attributed to infection brought by the blood-vessels or extending along the pancreatic ducts, since all these structures run in the interlobular tissues. But if the infection were propagated by these channels it should be diffusely distributed throughout the gland; and this is not the case in the earlier forms of the disease. It has been observed by all surgeons that the earlier forms of pancreatitis found at operation in connection with gall-bladder or duct disease involve only the head of the pancreas or perhaps only a portion of the head; and the "triangle" of duct-born infection described by Desjardins (see page 264) loses much of its interest when we consider that a *segmental pancreatic lymphangeitis* explains the limitation of the infection to the head of the pancreas in a much more rational manner. Thus *the distribution of the pancreatic inflammation corresponds to the lymphatic distribution*, which, as pointed out above, is irregularly *segmental*. It does not correspond to the duct distribution, which ramifies by dichotomous division from the main accessory ducts. If the infection were ductal in origin, the gland should be symmetrically involved. This point has been emphasized by Arnsperger.

The swellings of the head of the pancreas which are so frequently encountered by the surgeon must be different from the varieties of chronic pancreatitis described by the pathologist. Kehr, on clinical grounds, has surmised that such a difference exists. "Chronic pancreatitis," which is characterized by inter-

lobular or interacinar deposits of fibrous tissue, can be no more curable than chronic nephritis or cirrhosis of the liver. But it is characteristic of the pancreatic swellings associated with biliary disease that they subside with the disappearance of the biliary infection; such swellings therefore must be due to œdema, congestion, and absorbable infiltrates. The subsidence of such a

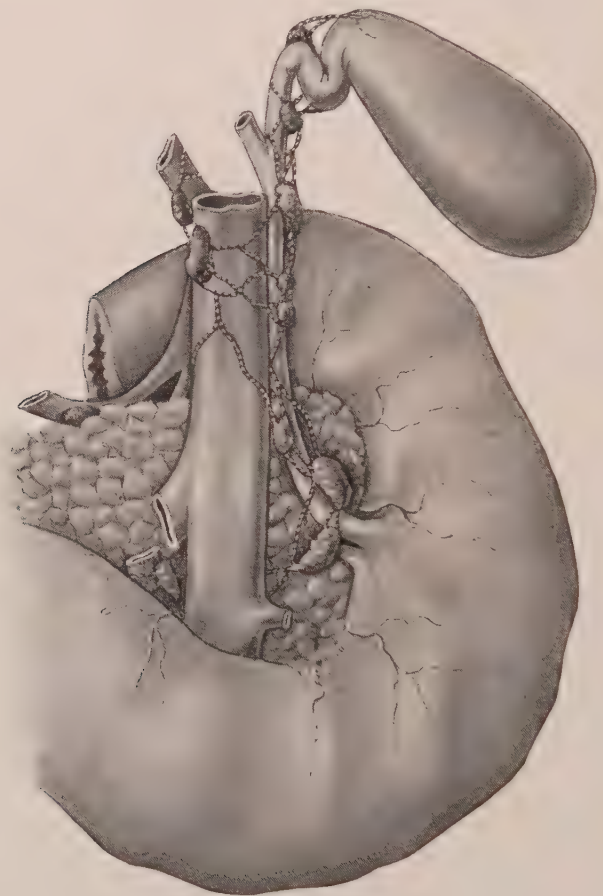

FIG. 22.—LYMPH-NODES OF THE BILE-DUCTS AND HEAD OF THE PANCREAS. (*Modified from Cunéo.*)

swelling in the head of the pancreas after cure of the primary infection is analogous to that which occurs in the treatment of the primary foci of lymphatic infection elsewhere in the body. In many cases of what we term pancreatic lymphangeitis it is possible to demonstrate the chain of infection: infected gall-bladder, enlargement of the cystic node, enlargement of the nodes

around the head of the pancreas, and swelling of the head of the pancreas, which corresponds to the regional lymphatic distribution. Since we have been looking for these lymph-nodes, it is remarkable how constantly they have been found in this condition. One node which appears to be especially constant in position and enlargement is situated just to the right of the choledochus where it passes beneath the duodenum.

We have spoken of this condition of pancreatic lymphangeitis hitherto chiefly in connection with gall-bladder lesions, because in such cases it is most easily recognized; but it is our belief that lymphatic infection from the pylorus and duodenum may play an important part in the pathogenesis of chronic pancreatitis. Not a few instances of the association of duodenal ulcer and pancreatitis are on record, while catarrhal duodenitis may also be a factor. It is possible that still other organs may at times furnish infection to the pancreas; but at present the biliary tract and the duodenum seem to be the chief sources of infection, on anatomical, clinical and pathological grounds.

A brief outline of the following case suggests that disease of the appendix may exert an influence on the pancreas.

Operation was performed for gangrenous appendicitis in a female child who had suffered with chills and high fever, the chills occurring two or three times in twenty-four hours. The appendix was removed. Chills and fever persisted and the child developed acute abdominal pain. A second operation was performed and a small abscess was found completely encapsulated in the mesentery of the ileum. Pancreatic lymphangeitis was present, the pancreas itself being greatly œdematous and infiltrated. Death occurred ten days after the second operation. Except for the pancreatic lymphangeitis above referred to, no lesion was found in the abdomen at autopsy.

The following are brief notes of a case in which Dr. E. G. Alexander operated, in the service of Dr. H. C. Deaver, Episcopal Hospital:

Adult male, aged 23, suffering with acute appendicitis, chills and high fever. The appendix was removed; no pus and few adhesions were found. Seventeen days after operation the patient had a marked chill, was reoperated on and a large mesenteric abscess drained. Death occurred five months after the second operation. Pathologic diagnosis was multiple abscess of liver, chronic fibroid peritonitis, post-operative peritonitis in region of appendix, acute splenic congestion, acute parenchymatous nephritis, suppurative cholecystitis and abscess of the head of pancreas.

In these cases it appeared that the source of the peripancreatic

and pancreatic inflammation was the infected appendix and that the path of metastasis was retroperitoneal and through the lymphatics along the cystic and common ducts.

It must be conceded that in many inflammatory conditions of the abdomen there is a retroperitoneal lymphangeitis which is fraught with the possibilities of injury to the pancreas since this lies almost directly in its path.

That this condition of pancreatic lymphangeitis has not been recognized hitherto at autopsy is no doubt to be explained in this way: the lesions are comparatively slight, and, even if the pancreas is examined microscopically, they can be confused easily with that autodigestion of the organ which ensues at once after death and so often renders examination of pancreatic tissue unsatisfactory; moreover the clinician has not called attention to the desirability of a search for minor pathological alterations. The disease is not itself immediately fatal; if it is relieved by the operative treatment instituted the pancreas may return to its normal state; while if unrelieved it will progress to true chronic interstitial (interlobular) pancreatitis, with deposition of fibrous tissue.

At the present time the *diagnosis* can be made only by the surgeon who palpates and inspects the gland during operation. The *symptoms* are those of the primary disease—cholecystitis, cholelithiasis, etc.; and the *treatment* involves only proper treatment of the causative lesion.

The following case is cited to show the extent to which the disease may progress. The pathology is too far advanced and the ætiology too obscure to prove its lymphatic origin in this instance. The glandular involvement, however, is similar in many respects to that observed by us in a considerable number of earlier cases where the considerations just mentioned lead to the belief in a lymphatic origin.

D. G., aged thirty-seven years. Admitted to the German Hospital, January 6, 1912. Died January 19, 1912.

Chief complaint, cramps in upper abdomen, radiating into flanks and to right side of back.

Family History.—Mother died of tuberculosis; otherwise negative.

Personal History.—Tea, coffee, and tobacco in excess; alcohol in moderation; denies venereal diseases. In March, 1910, he was admitted to the Pennsylvania Hospital suffering with pain beneath the right costal margin radiating to the back. The pain was worse two or three hours after eating.

Constipation was a marked symptom, and he had lost considerable weight. At operation cholecystitis was found and cholecystostomy performed. In July of the same year he was again operated upon for recurrence of symptoms and cholecysto-duodenostomy performed. In June, 1911, he was again in the hospital with the same symptoms plus jaundice, chills and fever. Cholelithiasis was diagnosticated, but he was not operated upon. In November, 1911, after intermittent attacks resembling those of stone in the common duct he came to the German Hospital and was again operated upon. Chronic pancreatitis, chronic cholecystitis, and a stone in the common duct were found. The stone was forced into the duodenum. The cholecysto-duodenostomy opening had become obliterated; cholecysto-duodenostomy performed. Recovery was uneventful, and he was improved for a few weeks, when his trouble recurred. More recently he has been having chills, fever, and jaundice.

Physical Examination.—A poorly nourished man of nervous appearance. Skin moderately jaundiced. Head and chest negative.

Abdomen: Scar of old incision through upper right rectus. In this region and in the epigastrium there is marked tenderness on pressure, but no rigidity or mass.

Urine shows a very faint trace of albumin, and a few hyaline casts, otherwise negative.

Blood: Hæmoglobin, 73 per cent.; erythrocytes, 4,050,000; leukocytes, 7,300 polymorphonuclear neutrophiles, 73.5 per cent.; lymphocytes, 26 per cent.; eosinophiles, 0.5 per cent.; coagulation time, 8 minutes.

Stool: Dark green and practically non-odorous; fluid; neutral reaction; a trace of bile, occult blood positive to benzidine and guaiac tests. Azotorrhœa and steatorrhœa marked.

Cammidge reaction positive.

Operation, January 13, 1912.—Dr. Deaver. Ether anæsthesia. Excision of old scar in upper right rectus region; adhesions of stomach and right lobe of liver to incision; hepatic flexure of colon adherent to right lobe of liver; great omentum adherent to lesser omentum; small whitish tubercles found on small intestines; some adhesions between ascending colon and parietal peritoneum; liver covered with a few whitish nodes which are subserous; old cholecysto-duodenostomy still patulous; head of pancreas enlarged and hard; felt semicystic; foramen of Winslow, though occluded, was forced open; small glandular enlargement at junction of the supra- and retroduodenal portions of the common duct; head of pancreas opened by inserting scissors and withdrawing them opened (Hilton's method). Much hemorrhage followed this; was controlled by a piece of selvage gauze and one suture of iodine gut; duodenum incised and old cholecysto-duodenostomy proved patent; much bile-stained material escaped from opening; head of pancreas seemed to be obstructing the duodenum posteriorly from pressure; duodenum closed with catgut baseball suture and a Lembert of linen thread; pancreas at site of incision sutured to parietal peritoneum; one cigarette drain anterior to gastrohepatic omentum; one rubber tube at foramen of Winslow; another rubber tube anterior to this and wound closed in layers.

Following the operation there was profuse brownish drainage from the wound. The jaundice disappeared. He became more emaciated and gradually weaker and died January 19, 1912.

Autopsy through the incision permitted removal of the pancreas and adjacent tissues. The head of the organ was somewhat smaller than normal, and appeared necrotic. The selvage gauze in the pancreatic wound had ulcerated into the duodenum at the site of the ampulla of Vater. A fistula existed at the site of the exploratory incision into the duodenum. A striking feature was the occurrence of many enlarged lymph-nodes along the upper margin of the pancreas. A few were present along the lower border, and in relation to the head both anteriorly and posteriorly where embraced by the duodenum.

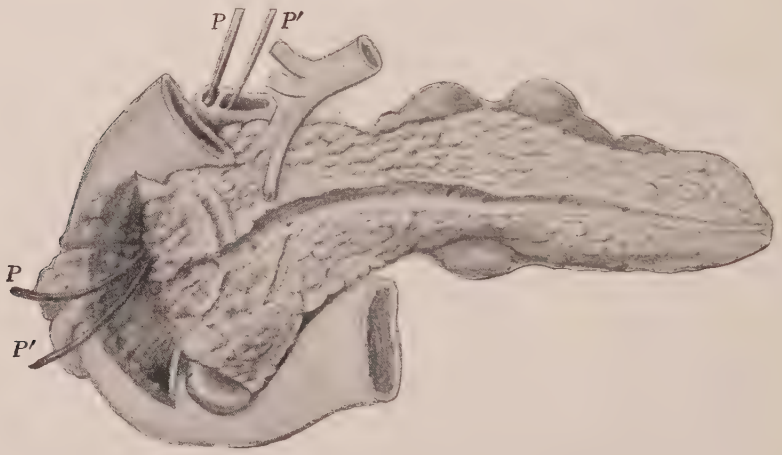

Fig. 23.—Anterior View of Pancreas, with Duct of Wirsung opened throughout its Length and a Window in the Duodenum.

P P, Probe from cystic duct through gall-bladder and cholecysto-duoduodenal anastomosis. *P′P′*, Probe from dilated common duct into duodenum through ampulla of Vater. (Case of D. G., page 320.)

Microscopically the tail of the pancreas was the seat of a moderate grade of interlobular sclerosis. The parenchyma was mostly degenerated, probably due chiefly to autodigestion. The islands of Langerhans were intact. The head of the organ showed marked interlobular pancreatitis and an acute suppurative process superimposed. The parenchyma was necrotic. The islands of Langerhans persisted but were involved in the acute necrotic process. A section made through a small villous patch at the junction of the gall-bladder and duodenum (at the cholecysto-duodenostomy opening) showed a marked glandular proliferation which had begun to invade the deeper tissues. The process had not extended more than 0.5 cm. from its starting-point, and no metastasis could be demonstrated. The lymph nodes showed chronic and acute hyperplastic lymphadenitis.

Chronic Interstitial Pancreatitis.—Attention was first prominently drawn to this condition by Mayo Robson in 1900. It is infectious in origin; and as we have pointed out in the preceding pages, the gall-bladder and bile-ducts are the chief primary foci.

Of seventy-nine patients in the German Hospital with chronic pancreatitis, seventy-two (91 per cent.) showed evidence of infection in the bile-passages; forty-two (53 per cent.) had gall-stones, and in thirty (38 per cent.) there was non-calculous inflammation.

W. J. Mayo reported 359 cases of pancreatitis, 86 per cent. of which were accompanied by gall-stone disease. Pancreatitis in some form was present in 7 per cent. of all patients where gall-stones were found in the gall-bladder; and in 27 per cent. of cases of stone in the common duct marked pancreatitis was present. This is in favor of the view held by Maugeret, and referred to at page 267, that the occurrence of pancreatitis results in stenosis of the bile-ducts, with resulting arrest of calculi in the common and hepatic ducts; but it also indicates that bile-duct infection predisposes to pancreatitis. A vicious circle is formed in this way.

Pathogenesis.—The *avenues of infection* in pancreatitis have been sufficiently discussed (page 259). From all that is known, we believe it is highly probable that in the vast majority of cases of chronic pancreatitis the infection occurs through lymph-channels and that the earliest stage of the disease is a pancreatic lymphangeitis.

Predisposing Causes. *Age.*—The age at which pancreatitis is said to occur depends upon whether the reported cases are from operative or autopsy records. This is readily demonstrated by comparing Opie's cases with ours.

	Opie.	Deaver.
Under 30 years	3 cases	4 cases (non-calculous)
30 to 40 years	2 cases	11 cases
40 to 50 years	9 cases	11 cases
50 to 60 years	11 cases	8 cases
Over 60 years	5 cases	0 cases
Total	30 cases	34 cases

More than two-thirds of Opie's cases (autopsy records) oc-

curred between the ages of forty and sixty. In our series, all but one being operative cases, two-thirds occurred between the ages of thirty and fifty.

Sex.—In thirty-seven of our patients with chronic pancreatitis without gall-stones, twenty-two were males and fifteen females. Opie reported seventeen males and thirteen females among thirty patients. Taken in conjunction with the well-known preponderance of gall-stone disease in women, these figures indicate that in men there frequently must be some other source of infection, probably the pylorus and duodenum.

Pathology.—Opie divides cases of chronic interstitial pancreatitis into two classes, *interlobular* and *interacinar*, with another form of degeneration, *lipomatosis*, common to both classes, and in each case the latest stage of the disease. While both kinds of pancreatitis consist essentially in the deposition of newly formed connective tissue, the difference between them is due to the position in which the fibrous tissue is laid down, and this pathological difference corresponds to differences in ætiology and symptomatology.

From a surgical standpoint chronic interlobular pancreatitis is the only form of interest, as interacinar pancreatitis is not amenable to operative treatment.

Chronic Interlobular Pancreatitis.

The interlobular connective tissue is not well marked in the normal pancreas, consequently the lobules are not sharply defined. In interlobular pancreatitis, however, these bands of connective tissue become greatly increased in size and density, accentuating the lobulation. A "typical" case of chronic interlobular pancreatitis has the following characteristics: In most cases, at least early in the disease, the head of the pancreas is the part affected. The probable reasons for this we have already stated in the discussion on pancreatic lymphangeitis. Only very late is the entire pancreas involved. The portion of the pancreas affected is enlarged and hard with a nodular surface; on section, tense bands of fibrous tissue transverse the cut surfaces in relatively the same manner as the normal interlobular framework, but the increase in amount and density results in the formation of well-marked lobules. From this classical type, readily recognizable at operation or autopsy, all grades

of interlobular change occur down to those cases in which the pancreas shows no macroscopic evidence of disease.

Microscopical Examination.—The most striking feature of this form of pancreatitis is the escape of the islands of Langerhans from any involvement in the sclerotic process. It is only very late in the disease, if at all, that they are involved. The deposition of connective tissue is most marked in the interlobular tissue, although, as the disease advances, there is a certain amount of invasion of the parenchyma with the replacement of normal parenchymatous cells by fibrous tissue. Later, this replacement may become so well marked that nothing of the parenchyma is left but the islands of Langerhans, which appear proportionately in greater numbers on account of the shrinkage of the connective tissue. Even at this late stage the islands of Langerhans are intact and functionating, as is shown by the non-interference with carbohydrate metabolism. When glycosuria does occur, as it may occasionally, late in the disease, it is due to interference with the blood supply of the islands of Langerhans from the pressure of the contracting fibrous tissue on the arteries.

Lymphoid cells, plasma cells and eosinophiles are numerous in the newly formed connective tissue, an evidence of active inflammatory change.

If this form of pancreatitis was caused primarily by obstruction or infection of the ducts, the parenchyma surely would be involved very early in the disease, since this is the active secreting portion of the pancreas connected with the excretory ducts. As already stated, however, parenchymatous changes occur late in the disease, the interlobular connective tissue being affected long before the parenchyma, and the islands of Langerhans remaining unaffected.

A certain amount of obstruction of the pancreatic ducts may result in this form of pancreatitis, from pressure by the interlobular fibrotic changes. In this way a condition is produced somewhat similar to that seen in experimental obstruction of the pancreatic ducts, which in animals results in a form of interlobular pancreatitis without involvement of the islands of Langerhans.

Chronic Interacinar Pancreatitis.

The deposition of newly formed connective tissue may take place within instead of between the lobules of the gland, with the

result that the parenchyma is largely replaced by fibrous tissue, which is *more or less evenly distributed throughout the substance of the pancreas* without accentuating the lobulation. This form of pancreatitis frequently is associated wth sclerotic changes in the arteries; and as a consequence the nutrition of the islands of Langerhans is affected early, and diabetes often results. The process sometimes seems to start in the islands of Langerhans. Sixty-seven of ninety cases of diabetes showed interacinar pancreatitis, and in sixty-five the arteries of the pancreas were sclerotic (Cecil). As a consequence there is interference with the carbohydrate metabolism and the appearance of glycosuria (pancreatic diabetes). This condition contraindicates operation; therefore interacinar pancreatitis is of secondary importance in a work on surgery.

Lipomatosis.

This condition consists in the deposition of large amounts of fat in the connective tissue. It may occur in either form of pancreatitis. Its cause and significance are not understood and while it does occur in fat people, obesity is not a necessary factor in its production, as it also occurs in patients who have lost a good deal of weight.

Symptoms and Diagnosis.—The extremes of opinion in respect to the diagnosis of chronic interstitial pancreatitis are represented by the pessimism of Opie and the optimism of Robson and Cammidge. The former says that "chronic pancreatitis is rarely accompanied by such definite symptoms that its recognition is possible during life" (page 243), while the latter hold that "from the information obtained from a careful examination of the patient, a knowledge of the history of the case, and the results of a chemical and microscopical examination of the excreta, a correct opinion may be formed in a large majority of instances." Our feeling in this matter is midway between the two extremes. The diagnosis has been made often enough to demonstrate that it is not too difficult to attempt; but it is equally true that our present criteria are too uncertain and inconstant to warrant a claim for great accuracy. There are no pathognomonic symptoms. Chronic pancreatitis is so often associated with disease of surrounding organs that it is difficult to separate the symptoms due to the accompanying disease and those due to the pancreatitis. Of course disease of the bile-ducts, particularly the common

duct, suggests the probable presence of pancreatic disease, although the symptoms referable to each condition cannot be differentiated.

The factors to be considered in making a diagnosis are:

1. The clinical history.
2. The physical examination.
3. Special tests designed to show disturbance of pancreatic function.

The following symptoms are those obtained in the analysis of a series of thirty-eight cases of chronic pancreatitis (under the senior author's care) in which there were no gall-stones present at the time of operation. As twelve of these patients had demonstrable changes in the gall-bladder, there may be a certain admixture of symptoms referable to involvement of the bile-passages.

Another point to be remembered is that these thirty-eight cases represent patients that underwent operation, consequently one would expect symptoms to be more marked than in a series of cases studied post-mortem. This point may serve as a possible explanation of the extremely divergent views on the possibility of diagnosis, the pathologist's view being advanced by Opie and the surgeon's by Robson.

Previous History.—Particular attention should be paid to a history of previous gastro-intestinal trouble or habits of eating and drinking likely to cause it; also to the occurrence of any of the infectious diseases habitually followed by cholelithiasis. If disease of the bile-ducts and gall-bladder has preceded the pancreatic inflammation, the early history presents the symptoms of that disorder, with perhaps frank attacks of biliary colic.

The relation of age and sex to chronic pancreatitis has been discussed (page 323).

Symptoms.—The onset of symptoms was sudden in twenty-five cases and gradual in thirteen; but these figures refer especially to exacerbations, premonitory symptoms being present in the majority of instances.

The symptoms usually associated with chronic pancreatitis are pain, nausea and vomiting, icterus, fever and loss of weight. These are common to various upper abdominal diseases and do not of themselves, even when all are present, constitute a characteristic symptom-complex. Mayo Robson and Cammidge attach

importance to symptoms only when they are accompanied by diagnostic changes in the urine and fæces, as demonstrated by chemical analysis. It is on these examinations more particularly than on the history and physical examination that they base their claim for the possibility of accurate diagnosis.

Pain is a leading and most constant symptom. It was present in all but three of our cases. This, of course, represents operative practice. The general practitioner must see many patients in whom pain is absent or so slight as to be negligible.

The *character* of the pain is not constant. It varies from a dull ache with a sense of fullness or oppression in the epigastrium to sharp lancinating pain like gall-stone colic. In our series it was severe in twelve cases, moderate in twenty-one, absent in three and not mentioned in two histories. Eleven patients had *distinct attacks of colic* and in the majority of these the gall-bladder was diseased. In one instance several stones were passed before operation although no stones were found when the abdomen was opened. It is probable therefore that colicky pain seldom occurs in chronic pancreatitis unless the bile-passages are involved. Colic from the passage of pancreatic calculi is indistinguishable in character from biliary colic, but the pain of chronic pancreatitis is the result of inflammatory changes and is not a colic. Yet Archibald, having observed on several occasions attacks of colic in patients in whom at operation no biliary lesions were demonstrable, suggests that these colicky pains may have been caused by retrojection of bile into the pancreatic duct, his idea being that during the long periods of fasting which patients with chronic pancreatitis undergo, the back pressure of bile is so augmented as to cause it to enter the pancreatic duct, though it is insufficient to force the sphincter of Oddi, except when this may become relaxed by physiological stimulation when chyme enters the duodenum (Vol. I, page 39). Archibald adds that the condition of fasting referred to above, as being likely to leave the sphincter closed, is especially frequent in chronic alcoholics, among whom this form of pancreatitis is so frequent.

According to Sailer the administration of a large amount of glucose to test the assimilation limit is particularly distressing to patients with chronic pancreatitis.

Michel quotes Desjardins, Vautrin and Dieulafoy as stating

that insidiousness is the dominant characteristic of the pain of chronic pancreatitis.

The site of pain varies as does the character. It was in the epigastrium in seventeen cases in our series, beneath the right costal margin in fifteen cases, beneath the left costal margin in one case, and in the lumbar region in two cases.

Radiation from the original site to the epigastrium occurred in five cases, to the back in nine cases, to the right costal margin in eight cases, to the left costal margin in one case, to the right shoulder in nine cases and to the left shoulder in two cases. Mayo Robson says that the attacks of pain have been mistaken for those of angina pectoris.

No definite relation to eating, drinking, or any particular food could be determined, a point of possible value in the differential diagnosis from gall-stone disease, gastric or duodenal ulcer.

From these figures it is evident that the pain occurring in the course of chronic pancreatitis is not constant in character, position or radiation, nor distinctive enough in any way to differentiate it from the pain of other abdominal diseases.

Nausea and Vomiting.—Twenty-one patients gave a history of vomiting at some period of the disease and ten more were nauseated but did not vomit. In four of Opie's cases, vomiting was so persistent and prolonged that the gastro-duodenitis causing it was considered the starting-point of an ascending infection of the pancreatic duct resulting in chronic interlobular pancreatitis. In none of our cases was the vomiting persistent, but the frequency with which it occurred indicates its importance as a symptom in the advanced type of the disease. The character of the vomiting is not distinctive, the vomitus consisting of stomach contents, bile and mucus.

Eructations of gas are common in this as in other diseases of the upper abdomen. They are mentioned as distressing in twelve cases.

Ten patients suffered from vomiting before the onset of pain, due in all probability to associated conditions in the upper abdomen.

Jaundice.—Eleven patients were jaundiced on admission, thirteen gave a history of previous attacks, and fourteen never had been jaundiced.

Jaundice is not a symptom of great value in differential diagnosis because of the various factors capable of causing it. Gallstones or inflammation of the bile-passages so frequently accompany pancreatitis that this explanation of the occurrence of jaundice is acceptable in a large proportion of cases and Opie considers it adequate in practically every case. However, the problem is not so simple. A certain number of cases of pancreatitis with jaundice occur without any demonstrable lesions in the biliary system. The relation of the common bile-duct in its lower third to the head of the pancreas offers a reasonable explanation under these circumstances. In about two-thirds of instances the lower end of the common duct passes through the substance of the head of the pancreas. The occurrence of jaundice in these cases is explained by the mechanical effect of swelling of the gland causing obstruction to the flow of bile. This factor also explains the persistence of jaundice after the passage of a stone from the common duct. The stone causes obstruction and inflammatory changes resulting in jaundice and pancreatic lymphangeitis. The latter in its turn causes swelling of the head of the pancreas, which by pressure on the common duct keeps up the obstruction after the stone has been passed.

The fact that in about one-third of instances the common duct passes behind and not through the head of the pancreas explains why in a definite proportion of cases of well-marked pancreatitis jaundice does not occur.

The degree of jaundice varies from a slight tinge to the "black jaundice" of the older writers, which was supposed to be diagnostic of malignant disease. Various observers have attempted to differentiate jaundice caused by pancreatitis from that due to other conditions, but without conspicuous success. Yet it seems to be a fact that jaundice from pancreatic obstruction is less subject to variation than that due to stone in the common duct. Michel says the characteristics of jaundice in pancreatitis are variable intensity at the onset, then progressive deepening without special pain. Kehr says that obstruction from a calculus lodged in the upper part of the common duct is accompanied by variations in intensity and by intermittence of jaundice because of the length and mobility of the common duct; but obstruction in the lower part of the common duct, particularly the ampulla, causes

continuous jaundice without great remissions. This latter variety, therefore, is very difficult to distinguish from jaundice due to pancreatic disease.

These observations simply prove that jaundice is a symptom upon which no great stress can be laid in making a differential diagnosis of upper abdominal lesions.

When jaundice is continuous and associated with rapid wasting and loss of strength, the clinical picture is that of carcinoma of the head of the pancreas. In five of our cases jaundice was continuous, in the others, intermittent. Intermittent jaundice, pain and fever may occur in the course of chronic pancreatitis and simulate stone in the common duct with Charcot's hepatic intermittent fever.

The *relation of pain to jaundice* in our cases was indefinite. Four times the onset of jaundice was not accompanied by pain. Pain preceding the onset of jaundice may be due to gall-stone colic, but as a rule the pain is less severe and not of a colicky character.

Loss of Weight and Strength.—Emaciation and loss of strength are fairly constant accompaniments of pancreatitis and always manifest themselves when the degree of sclerosis is sufficient to interfere with pancreatic function. The obstruction to the excreting ducts diminishes the amount of secretion reaching the intestine, resulting in imperfect digestion and mal-assimilation. Another factor is restriction of diet, voluntarily or by the advice of a physician, in the attempt to control the symptoms of indigestion. At times impairment of appetite is responsible for decreased intake of food, though loss of appetite is by no means a constant accompaniment of pancreatitis. It was mentioned seven times in our series. If jaundice is present biliary intoxication may be a factor in the production of emaciation.

Loss of weight was noted in twenty-one cases. The state of nutrition at the time of operation was poor in thirteen cases, good in ten, while six patients were obese; in nine patients the state of nutrition was not mentioned. Loss of strength follows loss of weight and was complained of by ten patients.

Very rapid loss of weight may take place in some instances, particularly when jaundice is a marked feature. Cachexia may be more rapid and extreme than in malignant disease. Moynihan reports a case where a patient lost 26 pounds in three months;

Mayo Robson reports patients losing 42, 55 and 110 pounds; Chauffard, a patient losing 77 pounds in six months; and Terrier, a patient losing 16 pounds in one month and 44 pounds in eight months.

These are, however, exceptions, and the usual case does not exhibit such marked wasting until a late stage of the disease.

Fever.—Fever is not such a prominent symptom in the prolonged course of chronic pancreatitis as most figures would seem to indicate. As a rule the temperature is normal. Hyperpyrexia is present only during exacerbations. Of our series five patients gave a history of chills and sweating. At the time of operation thirteen patients had a temperature between 99° and 100° F. In four it was between 102° and 103° F.

Bowels.—Constipation is the rule; nineteen of our patients suffered with chronic constipation and in thirteen constipation was a feature of the attacks. In only five was there a history of diarrhœa.

From this it must be seen that caution must be used in employing as an aid to diagnosis the classical description of "frequent bulky motions, pale in color, offensive, and obviously greasy." As stated by Robson and Cammidge, such stools are present only in advanced conditions. The stools are likely to be clay colored even when bile is present. If bile is persistently absent to laboratory tests the condition is more apt to be carcinoma of the pancreas than an inflammatory affection.

Diabetes.—Glycosuria indicates involvement of the islands of Langerhans in the sclerotic process. It is a rare symptom in interlobular pancreatitis and is then a sign that the destruction of the parenchyma has advanced to such an extent that the fibrous tissue has either encroached directly on the islands of Langerhans or has, by contraction, seriously interfered with their blood supply. Two cases of our series showed glycosuria. Sugar may appear in the urine during an exacerbation and clear up on subsidence of the inflammation. This is a threat of oncoming diabetes and should not be overlooked. Of equal significance is the presence of alimentary glycosuria, tested by the administration of sugar. Drainage of the infected biliary and pancreatic ducts may cause disappearance of glycosuria when it is associated with interlobular pancreatitis and is of recent development.

In interacinar pancreatitis the symptoms of true diabetes mellitus appear early in the course of the disease. Recognition of one of the various diseases usually associated with pancreatic diabetes, such as arterial sclerosis, taken in conjunction with the early appearance of sugar in the urine, usually enables a distinction to be made between this form of pancreatitis and that occurring in the interlobular type.

Physical Examination.—The physical examination rarely affords much positive information. It is of more value in excluding other conditions.

Tumor.—During exacerbations there may be epigastric tenderness and rigidity which completely mask the underlying condition. Rigidity was absent in the right hypochondrium in nine cases and over the epigastrium in three. There was moderate distention in three cases. In patients with thin abdominal walls the swollen head of the pancreas sometimes may be palpated between exacerbations of the disease. In three of our cases there was an indefinable mass to be felt. As a rule, palpation even of a considerably enlarged pancreas is impossible, as in most instances the pancreas is well covered by the adjacent organs.

Körte examined thirty cadavers with reference to this point. In twenty the pancreas was completely covered, while in ten there was some part covered with omentum only. In six there was ptosis of the colon with exposure of a portion of the head of the pancreas between the liver and colon; in two it was exposed in the median cleft of the liver; in the other two there was marked gastroptosis and the pancreas could be palpated directly beneath the gastrohepatic omentum. In palpating through the abdominal wall these slight exposures of pancreatic tissue rarely are sufficient to give definite results.

When a mass is demonstrable it may be referable to an enlarged gall-bladder or a cancer of the head of the pancreas as well as to chronic interstitial pancreatitis. Even when the abdomen is open the differentiation between carcinoma and chronic pancreatitis may be exceedingly difficult.

Enlargement of the gall-bladder has been observed in a number of cases, but in our cases it was not detected prior to the operation. The liver was noted as enlarged nine times.

Tenderness.—As most of our patients were operated on during

or just after some exacerbation of symptoms there was a degree of tenderness present in most of them. In eight no tenderness was elicited. In the remainder tenderness was found below the right costal margin in twenty; beneath the left in three; in the mid-epigastrium in eleven; over Mayo Robson's point[1] in three; and in one severe case it was general. Rigidity was observed in the right hypochondrium in nine cases and over the epigastrium in three. There was moderate distention in six cases.

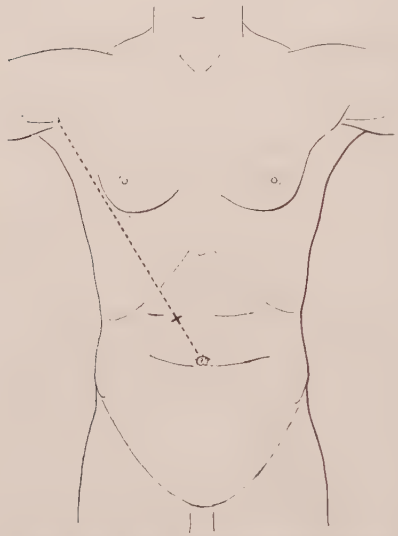

Fig. 24.—The "Pancreatic Point" of Desjardins and the "Pancreaticohepatic Area" of Chauffard and Rivet.

There is then no constant point or area to which tenderness is limited in cases of chronic pancreatitis, although various authors have described them. Desjardins describes a point 5–6 cm. above the umbilicus on a line drawn between it and the gall-bladder area, as the point on the abdomen representing the position where the duct of Wirsung enters the duodenum. Chauffard and Rivet, instead of describing a fixed point, name the area corresponding to the head of the pancreas the pancreaticohepatic area. This area occupies the lower part of an angle of 45 degrees formed by the mid-line and a line drawn up and to the right from the umbilicus (Fig. 24).

We think no dependence can be placed on these or similar points and areas of tenderness in differentiating conditions of the upper abdomen.

Blood Examination.—The blood frequently shows secondary anæmia which seldom is grave, but may become so if surgical intervention is delayed too long. In about one-half of our cases the hæmoglobin was below 80 per cent., and the erythrocytes below 4,000,000. A moderate leukocytosis was present during a

[1] This is a point "just above and to the right of the umbilicus."

few of the more acute exacerbations. Generally the numerical ratio of the leukocytes was unaltered.

Speciat Tests to Show Disturbances of Pancreatic Function.—The number and variety of laboratory aids to diagnosis show that none of them is infallible. The most important have been discussed at page 274.

Examination of Fæces.—The results of examinations of the fæces in our cases have convinced us that functional tests of pancreatic activity have not yet reached a stage which warrants much dependence upon them.

In ten of our cases the stools were clay colored. It is well known that this character does not depend so much upon the absence of bile as on the *presence of fatty acids* or of *fat itself*. The tests for stercobilin were positive in all of these instances, showing that bile was entering the intestinal tract. This is important and leads to the deduction that, since the liver is functionating, the failure to digest fats is due (gastric and intestinal disease having been excluded) to deficiency of the pancreas. An excess of fat so marked as to be visible to the naked eye was present in six cases, and an excess of fat was found on examination of the fæces in five cases. It remains to be seen whether the quantitative relations of the fat and its derivatives in the fæces will be of any great assistance. "In interpreting the results of an analysis of the fæces for fats one must take into account the other indications given by the clinical symptoms and an analysis of the urine and fæces" (Cammidge).

The determination of proteid digestion seems to be even less helpful. In very advanced cases undigested muscle-fibres may be found in the fæces, sometimes by the naked eye, but more often only by microscopic examination. Fitz says this occurs "only when there is extreme diminution of the pancreatic juice, and is significant only when gastric digestion is normal, when the diet contains no excess of meat, and when there is no diarrhœa" (Opie).

The determination of the activity of carbohydrate digestion is said to give valuable aid in arriving at a diagnosis of pancreatic disease. If by Müller's test no proteolytic enzyme is demonstrable in the fæces Schlecht considers it an indication that pancreatic juice is not reaching the intestine. Gross and Heiberg

use an alkaline solution of casein instead of the blood serum used in Müller's test. The amount of digestion is measured by precipitating the undigested casein with dilute acetic acid. This is a long and rather difficult test as it requires a clear filtrate of the fæces free from bacterial growth.

Lynch, Fedeli and Romanelli have also devised tests to determine the activity of carbohydrate digestion (page 277).

"These tests are of considerable value in showing serious interference with the digestive functions of the pancreas such as is met with in advanced cirrhosis of the gland and in cancer, but they do not distinguish the early stages of chronic pancreatitis which are most amenable to treatment" (Cammidge).

The *reaction of the fæces* in pancreatic disease may be markedly acid; normally there is an amphoteric reaction.

Examination of the Urine.—It is advisable to collect a twenty-four-hour specimen. Cammidge recommends a simple mixed diet for two or three days beforehand, rather than any special test diet, as the idea is to measure the capacity for digesting normal food. Routine examination includes tests for the following: "Albumen, sugar, urobilin, acetone, diacetic acid, the amount of ammonia nitrogen, the results of the pancreatic reaction, and the presence of calcium oxalate crystals microscopically" (Cammidge).

The amounts of urobilin, acetone, di-acetic acid and ammonia nitrogen are so variable and their significance so uncertain, that they have comparatively little value in diagnosis.

The "pancreatic" or Cammidge Reaction (page 278).—As is well known this reaction consists in the demonstration in the urine, when treated by a rather complex chemical procedure, of certain crystals of a definite morphology and certain solubility characteristics, but of unknown composition, although it is thought to be a derivative of pentose, probably an osozone.

Opinions differ as to the value of the Cammidge reaction. Cammidge's own claim (1912) for the improved or "C" reaction is that, "a positive result, although not by itself sufficient to make a diagnosis of pancreatitis certain, is suggestive of active degenerative changes of an inflammatory character in the pancreas. A negative reaction is against there being any active pancreatitis, but does not exclude cirrhosis of the gland, the result of past in-

flammation, or malignant disease of the pancreas with which a negative result is obtained in two-thirds of the cases."

The results in our own cases justify the statement that, while the reaction may be a valuable confirmatory sign of chronic or acute pancreatitis, it is neither pathognomonic nor specific. Its absence by no means denotes the absence of pancreatic involvement.

Of five cases of acute or subacute pancreatitis the Cammidge reaction was positive in three.

Of six cases of chronic pancreatitis without biliary involvement it was positive in one.

Of twenty-one cases of chronic pancreatitis with biliary involvement but no stones, it was positive in five.

Of twenty-seven cases of chronic pancreatitis with gall-stones, it was positive in seven.

This gives 27 per cent. of positive reactions in fifty-nine cases.

Of sixty cases in which the pancreas appeared to be normal to direct palpation at the time of operation, ten (16 per cent.) showed a positive reaction. These result. and those previously alluded to (page 279) do not indicate that the reaction is as trustworthy as it was hoped it would be.

In the last few years a great deal has been written about the Cammidge reaction. Opinions differ as to its value. Some consider it valueless as an aid to diagnosis, others consider it more or less useful and still others apparently depend on the results of the "pancreatic" reaction more than on any other factor. Remembering that the degree of favor in which the reaction is held varies, a practical division of observers may be made into two groups, those who favor its value in diagnosis and those who do not. The first group includes Barker, Bernouilli, Desjardins, Erdmann, Goodman, Hagen, Kehr, Krietnitz, Kinnicut, Labbé, Moynihan, Ochsner, Pilcher, Mayo Robson, Schwarz, Speese, Taft, Taylor, Vautrin and Watson. The second group is much smaller and includes Carnot, Cartier, Chauffard, Dreesmann, Körte, Lepine, Quénu, Seidel, Villard and Wilson. Reference to these reports will show the grounds on which the authors base their opinions. (See Table of References, page 345.)

The presence of *steapsin* (*lipase*) *in the urine* was noted by Opie and by Hewlett in some cases of pancreatic disease. Accord-

ing to Archibald the test is made by adding to a sample of the urine to be examined a definite amount of fatty substance which the ferment, if present, can split. Butyric ether is used, its cleavage yielding free butyric acid. A control experiment is made using some of the same urine, which for the control test has been boiled so as to destroy the ferment. If on titration a greater amount of acid is found in the test urine than in the control, it is evident that the fat-splitting ferment is present. As Archibald points out, a large margin of error must be allowed for, in making this test, which he considers of confirmatory value if positive, but of no use if negative.

Glycosuria.—Two of our cases showed glycosuria. This is a sign that appears late in interlobular pancreatitis and is of grave significance. Sugar may appear in the urine during exacerbations and disappear in the intervals. Alimentary glycosuria is another important indication of pancreatic insufficiency that should be looked for.

Calcium Oxalate.—The presence of the characteristic crystals of calcium oxalate is said to be a confirmatory sign of pancreatic inefficiency.

Lipuria and *maltosuria* in the urine are signs of no particular significance. The presence of *diastase* in the urine, in pancreatic lesions, has been referred to at page 280.

Examination of Stomach Contents.—This was made in twenty-four of our cases. In two-thirds of these there was diminution of both free hydrochloric acid and in the total acids. In no case was there a marked hyperacidity. The great number of conditions associated with subacidity of the gastric contents deprives these observations of any value. In other respects gastric analysis gave normal results.

Various methods of obtaining *duodenal contents* have been proposed from time to time, but none of them in our opinion has any practical application.

Diagnosis.—Undue prominence seems to be given in the symptomatology of chronic pancreatitis to the description and discussion of the various laboratory methods for determining interference with the function of the pancreas. This may be taken as an indication of the views on diagnosis. The confidence of clinicians in their ability to diagnose this condition is directly

expressed by their faith in the accuracy and reliability of the various tests for pancreatic function, particularly the Cammidge reaction, and, to a less degree, the examination of the fæces.

From the symptoms and physical signs, localization of the disease to the pancreatico-hepatic region almost always can be made, but in the differentiation of chronic pancreatitis form disease of the bile-ducts, or in the diagnosis of pancreatitis accompanying or resulting from gall-stone disease there always is a large amount of doubt, as to the possibility of accurate localization, in the minds of those clinicians whose experience with the Cammidge reaction has been unsatisfactory and disappointing.

The symptoms of chronic pancreatitis are, in nearly every instance, those of an associated lesion or set of lesions. This point is well illustrated in the classification of chronic pancreatitis by Cammidge into:

1. The Dyspeptic form.
2. The Cholelithic form.
3. The Mixed form: found in carcinoma, or in pancreatitis associated with circulatory disturbances.
4. The Diabetic form—the last stage in interlobular pancreatitis.

Although most cases of chronic pancreatitis or pancreatic lymphangeitis might be classified according to their symptomatology into these groups, such classification gives no clue to the differential diagnosis in the absence of faith in the specificity of the "pancreatic" reaction. As the classification indicates, the symptoms supposedly due to pancreatitis may also be caused by the conditions giving their names to the various groups.

When a patient has developed diabetes it is almost certain that there is some pancreatic lesion and it is equally certain that surgery can be of no further benefit. Fortunately there is never any doubt of the diagnosis.

There are no pathognomonic signs of chronic interlobular pancreatitis, the suggestive factor being the association of various symptoms and physical signs, which have been sufficiently discussed. We do not consider the positive diagnosis of chronic interlobular pancreatitis possible, as we have never seen a case, diagnosed clinically chronic pancreatitis, operated upon and demonstrated to have this condition as the primary and most important

lesion. It is true that the presence of pancreatitis either alone or in conjunction with other lesions may be strongly suspected, but the diagnosis cannot be made with the same degree of certainty as in gall-stones, duodenal ulcer or appendicitis. In a very few instances, none of which have come under our direct observation, marked emaciation showing some grave metabolic change, with intermittent constipation and diarrhœa, and large stools with much undigested fat have pointed quite directly to the existence of a pronounced pancreatic lesion. In the vast majority of cases, however, these symptoms are not sufficiently marked to attract notice.

The differentiation from gall-stone disease is then very difficult if not impossible in the greater number of patients. Fortunately treatment is the same and the patient does not suffer from the surgeon's inability to localize the various lesions causing the symptoms. The presence of pancreatitis may be considered highly probable if the patient has had symptoms referable to the bile-passages persisting over a long period.

In cases of chronic jaundice, the presence or absence of stercobilin in the fæces may aid greatly in the diagnosis. Even in advanced cases of chronic pancreatitis obstruction of the common duct is rarely absolute, consequently some bile escapes into the intestines. A stone in the common duct, unless it completely blocks the outlet, acts in a similar manner. On the other hand, carcinoma of the head of the pancreas, as a rule, causes absolute obstruction and allows no bile to escape; consequently stercobilin is not to be found in the fæces. Carcinoma of the bile-ducts, or of the gall-bladder causing secondary obstruction of the ducts may cause the same absolute occlusion and prevent the escape of the bile. The presence of stercobilin is of suggestive rather than of diagnostic importance. Very often after the abdomen is opened and the pancreas examined directly by sight and touch, it is impossible to diagnosticate carcinoma of the head of the pancreas from chronic interstitial pancreatitis localized to the same region.

Chronic cholangeitis without gall-stones, and chronic appendicitis of the type manifesting itself chiefly or solely by upper abdominal symptoms, may also be mistaken occasionally for chronic pancreatitis.

Prognosis.—It does not appear that the existence of chronic

pancreatitis as a complication of biliary tract disease materially increases the immediate mortality of operative treatment, unless the pancreatitis has passed beyond the curable stage. (See Table at page 112.) Nor does it seem that the expectation of life is materially less. In other words, the prognosis, both immediate and remote, in early cases of chronic pancreatitis is much the same as in diseases of the biliary tract, and varies with the complications present in the gall-bladder and bile-ducts.

Treatment. *Medical Treatment.*—In mild cases where the diagnosis is extremely uncertain, medical treatment should be continued if improvement occurs. It is not improbable that many of the so-called "stomach complaints," catarrh, etc., are mild cases of pancreatic lymphangeitis that end in recovery. That these patients may be benefited by a "cure" at one of the famous springs is highly probable. But too great delay in resort to surgical measures is mistaken policy, especially when the diagnosis of the usual underlying biliary complaint is certain, since chronic pancreatitis, when once it has reached the stage of fibrous deposit, is incapable of being cured, although possibly the still further progress of the disease may be arrested by timely operation.

Surgical Treatment.—Surgical treatment of chronic pancreatitis aims at the accomplishment of three things:

1. Removal of the underlying cause.
2. Prevention of further involvement of the pancreas.
3. Cure of the pancreatic disease present.

Removal of the underlying cause is not always easy, as it is no easy matter to determine it in all instances. When, however, there is evidence of infection of the biliary system, meeting these conditions is sufficient to do away with the pancreatic disease in the majority of instances.

In cases of biliary infection, calculous or otherwise, operative treatment involves more or less prolonged drainage of the biliary tract, and this furnishes the correct mode of meeting the pancreatic condition.

The easiest and most advantageous method of providing drainage is by cholecystostomy. If the gall-bladder is greatly diseased and full recovery doubtful, cholecystectomy is indicated. Drainage of the common duct should then be established.

There is no doubt that the pancreas may be drained through

the opening in the common duct. In certain cases the discharge is peculiarly irritating to the skin, often causing excoriation. The presence of pancreatic ferment is demonstrated by the digestion of blood-serum and starch in alkaline solution. This we have done to prove that draining the common duct also drains the pancreas. These findings were obtained in cases where the patency of the outlet of the common duct was insured by the passage of a good-sized gall-stone explorer through the ampulla into the duodenum. When the pancreatic duct does not open into the sinus of Vater, this avenue for drainage of the pancreas is not open; but the operation is productive of good in that the primary focus of infection in the gall-bladder is abolished, and the accompanying lymphangeitis of the pancreas cured.

At times when the closure of the common duct is complete and likely to be lasting, a cholecystenterostomy may be best. Robson and Kehr are of the opinion that drainage by cholecystenterostomy will cure 97 per cent. of cases of chronic pancreatitis. Much as we dislike to disagree with such eminent authorities, we are still partial to external drainage. As a general rule this permits subsidence of the swelling of the pancreas and a re-establishment of the functions of the ducts. This drainage should be maintained from *four to eight weeks at the very least*, or until the bile becomes sterile. Often it is difficult to keep the fistula from closing too soon, but in cases of marked pancreatic disease the sinus may continue to discharge bile for months. This should not discourage either the patient or the surgeon, provided the presence of bile in the fæces, even in slight amount, can be ascertained. Vautrin had to wait nine months in one case for the choledochus to become permanently patent and for the biliary fistula to close; Kehr, Körte and others have had to wait three or four months for the sinus to close. The prolonged drainage is beneficial; many patients have had recurrence of symptoms when the sinus closed and it has had to be reopened. It is our practice in cases of chronic pancreatitis, always to pass a sound through the common duct into the duodenum, to make certain that the passage is permeable. Only when at operation the obstruction of the common duct is very marked, and the head of the pancreas very hard, do we think it proper to resort to cholecystenterostomy even as a secondary operation. Just how long the cholecysto-intestinal anastomosis

remains patulous is a question, for in many cases reoperation has demonstrated that the opening has closed. Cholecysto-duodenostomy is an operation having a higher mortality than cholecystostomy, which fact should be borne in mind when deciding the operation to be adopted in the individual case.

The problem of the treatment of pancreatitis when the gall-bladder seems to be practically normal is a more difficult one. In many of these cases close observation shows that the gall-bladder exhibits some interstitial thickening and possibly opacity of the serous coat. Such gall-bladders frequently contain thick tarry looking bile, many times containing micro-organisms, perhaps anaërobic. These are probably cases of mild hepatic infection, with inconspicuous involvement of the gall-bladder. Drainage is as efficacious in these as in cases which show more advanced pathological changes. In some of these cases the origin of the trouble is in pyloric disease, and the separation of pyloric adhesions, gastro-enterostomy, or other operation may be indicated. When no other lesion can be discovered Vautrin advocates attacking the pancreas directly, especially urging drainage of the retropancreatic tissues after exposing this region by mobilization of the duodenum (Fig. 25). In one case he used the thermo-cautery to liberate the common duct from its position within the dense pancreatic head.

The means employed to remove the underlying cause of pancreatitis are also best adapted to prevent extension of the process and to cause a restitution to normal, so far as this is possible, of pathological lesions already present. In pancreatic lymphangeitis, proper drainage reduces to a minimum the probability of an interstitial fibrotic change being superimposed upon it.

As we have already indicated, we think cholecysto-duodenostomy rarely or never is indicated for the treatment of chronic pancreatitis. If the common duct is patulous, it is extremely improbable, as indicated by Archibald's experiments, that this operation diverts the bile from its natural channel; and it is our belief that it should never be employed as a primary operation. In those very rare cases of chronic pancreatitis where a biliary sinus persists indefinitely after drainage of the gall-bladder, this operation may be employed with propriety, and in most cases with complete relief of symptoms. But the danger of an ascending cholange-

itis always exists, and the operation should not be adopted without mature consideration. If the gall-bladder has been inadvisedly removed at a previous operation, one of the other methods of restoring the continuity of the bile-passages must be employed (page 124).

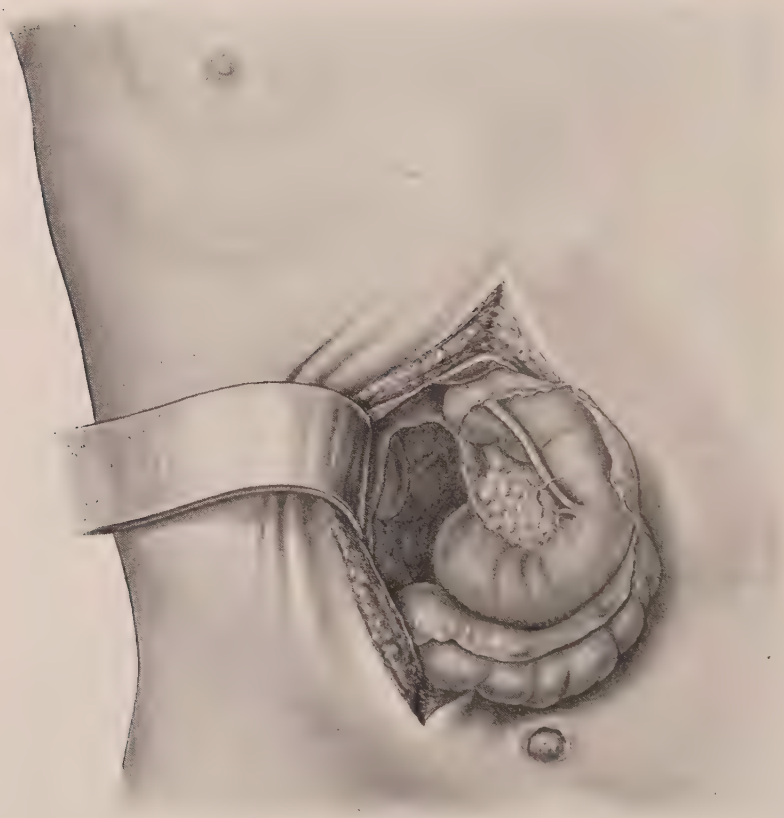

FIG. 25.—EXPOSURE OF THE HEAD OF THE PANCREAS AND COMMON BILE-DUCT AFTER MOBILIZATION OF THE DUODENUM. (*After Guibé.*)

The whole question of treatment for chronic pancreatitis resolves itself into an earnest endeavor promptly to treat upper abdominal disease by appropriate surgical means when medication has been ineffectual or when the symptoms closely point to a pathological condition not amenable to the ordinary therapeutic measures.

BIBLIOGRAPHY.

Archibald: Maritime Med. News, 1910, xxii, 105.
Archibald: Canadian Med. Assoc. Jour., 1912, ii, 557.
Arnsperger: Münch. med. Woch., 1911, lviii, 729.
Barbour: Archiv. Int. Med., 1911, viii, 662.
Barker, E.: The Lancet, 1910, ii, 1264.
Bartels: Arch. f. Anat. u. Physiol., Anatom. Abt., 1904, S. 299; 1906, S. 250; 1907, S. 267.
Bernouilli: Correspondenzblatt für Schweizer Aerzte, 1910, xl, 297.
Cammidge: The Lancet, 1911, ii, 1494.
Cartier: Thèse de Paris, 1909.
Carnot: Thèse de Paris, 1898.
Cecil: Jour. Exp. Med., 1911, xiv, 500.
Chauffard and Rivet: III Congr. Internat. de Chir., 1911, p. 455, quoted in this place by Michel.
Deaver, John B.: Jour. Amer. Med. Assoc., 1911, lvi, 1079.
Deaver, John B.: Jour. Am. Med. Assoc., 1913, i, 1.
Deaver and Pfeiffer: "Pancreatic Lymphangeitis," Amer. J. Med. Sc., 1912, i, 473.
Desjardins: Thèse de Paris, 1898.
Dreesmann: Münch. med. Woch., 1909, lvi, 708.
Franke: Deutsch. Zeit. f. Chir., 1911, cxi, 539.
Giordano, D.: Congres de la Soc. Internat. de Chir., 1911, iii, 557.
Goodman: Annals of Surg., 1909, xlix, 183.
Hagen, W.: Beitr. z. klin. Chir., 1909, lxi, 750.
Hewlett: Jour. of Med. Research, 1904, n. s. vi, 377.
Kehr: Münch. med. Woch., 1909, lvi; 1062.
Kinney: Amer. Jour. Med. Sc., 1910, cxl, 878.
Kinnicut: N. Y. Medical Record, 1909, lxxv, 589.
Körte: Med. Press and Circ., 1911, i, 512.
Krietnitz: Arch. f. Verdauungskr., 1909, xv, 53.
Labbé and Vitry: Bull. Soc. Méd. des Hôpitaux, Paris, 1910, xxix, 829.
Mayo: Collected Papers, 1911, Phila., 1912, pp. 141; 151.
Mayo: Surg., Gynec. and Obstet., 1908, vii, 607.
Michel: Congres de la Soc. Internat. de Chirurgie, 1911, iii, 447.
Morris: American Med., 1905, x, 95.
Moynihan: British Med. Journ., 1909, ii, 936.
Ochsner: Journ. Amer. Med. Assoc., 1910, liv, 1776.
Opie: Osler's Modern Medicine, Phila., 1908, vol. v, p. 621.
Opie: Diseases of the Pancreas, 2nd Ed., Phila., 1910, p. 198 (Lipase in urine).
Pilcher: Annals of Surgery, 1910, i, 89.
Quénu: Journal des Practiciens, 1910, xxiv, 403.
Roper and Stillman: Arch. Int. Med., 1911, vii, 252.

Robson and Cammidge: "The Pancreas, its Surgery and Pathology," Phila., 1907.
Sailer: Amer. Jour. Med. Sc., 1910, cxl, 330.
Schwarz: Wien klin. Wchnschr. 1909, xxii, 297.
Seidel: Verhandl. d. Deutsch. Ges. f. Chir., 1909, xxxviii, 158.
Speese and Goodman: Amer. Jour. Med. Sc., 1909, cxxxvii, 107.
Taylor, A. E.: Lancet, 1906, i, 1818.
Terrier: Bull. Soc. Chir., Paris, 1906, xxxii, 164.
Thomson, G. Y.: Lancet, 1911, ii, 221.
Vautrin: Rev. de Chir., 1908, xxxvii, 589.
Watson: Lancet, 1908, ii, 1519.
Whipple, Chaffee and Fisher: Bulletin of the Johns Hopkins Hosp., 1910, xxi, 339.
Wilson, L. B., Willis, B. C., and McGrath, B. F. (et al): Trans. Ass. Am. Phys., 1910, xxv, 192.

Pancreatic Calculi.

In contradistinction to gall-stones, pancreatic calculi are of very infrequent occurrence. Johnston collected thirty-five cases in 1883, and by 1904 Lazarus found only a total of fifty-seven cases on record. Little that is definite is known of the factors which cause their formation. This aspect of the question, as well as certain aspects of the clinical pathology of the subject, have been discussed at page 268.

Symptoms.—As is the case with all other chronic diseases of the upper abdomen, pancreatic calculi may exist for years without causing any definite symptoms or physical signs. Cases have been reported frequently where stones previously unsuspected have been found at autopsy. There is nothing distinctive in the group of symptoms usually associated with pancreatic calculi—pain, nausea and vomiting, jaundice, glycosuria, steatorrhœa, azotorrhœa and digestive disturbances.

Pain.—This varies in severity from an indefinite dull ache or sense of pressure, to attacks of acute colic scarcely to be distinguished from biliary colic. And just as "biliary colic" may be caused by violent peristaltic contractions of the gall-bladder and ducts, in the absence of all calculi; so it is probable that "pancreatic colic" does not always depend on the presence of calculi in the pancreatic ducts. In many cases of acute pancreatitis there is a history of previous

attacks of epigastric pain, which may have been due to small hemorrhages or possibly to violent peristaltic contraction of the pancreas. In situation and radiation the pains usually are indistinguishable from those accompanying biliary colic, as is evidenced by the fact that pancreatic calculi have been so frequently diagnosed "gall-stones." The persistence of colic, after an operation for gall-stones, may be due to calculi in the pancreas (cases of Körte and Kümmell). This, however, is a very rare event. The pain is said to be more severe in the left epigastrium and to radiate to the left scapular region instead of the right; but in other cases the pain starts in the back and radiates around the side or straight through the body (Kinnicutt). *Nausea* and *vomiting* are constant accompaniments of the severe attacks of pain, as is the case in biliary colic. *Rigors* and *collapse* may also occur. Subsequent to these attacks of pain, calculi or fragments of calculi may be recovered from the fæces. Analysis of these stones shows the usual composition of pancreatic calculi; as noted at page 269, they consist largely of calcium carbonate and phosphate. A stone that has been lodged long in the ampulla of Vater may become coated with bile-salts and biliary coloring matter until it resembles a gall-stone, but the nucleus of the stone presents the characteristic composition. Such a stone may set up *jaundice* from obstruction of the common duct. This is not an uncommon symptom in connection with pancreatic lithiasis. It has the usual characteristics of obstructive jaundice. If the stone is passed jaundice is temporary, but if the stone lodges the jaundice remains and probably increases. Jaundice may be due not to blocking of the duct by a stone in the ampulla of Vater, but to an associated cholelithiasis or bile-duct infection, or to obstruction from pressure by the head of the pancreas, since in association with pancreatic lithiasis there always is a certain amount of pancreatitis. Occasionally this is of extreme degree; then there may be symptoms referable to the gastro-intestinal tract resulting from pancreatic indigestion, with steatorrhœa and azotorrhœa and other characteristic changes found in association with chronic interstitial pancreatitis.

Intermittent or permanent *glycosuria* occurs in about half of the cases, and is to be attributed to involvement of the islands

of Langerhans in the sclerotic process. Alimentary glycosuria may be present occasionally. Flatulence, indigestion, loss of weight, etc., are to be referred to the accompanying pancreatitis rather than to the calculus.

Robson and Cammidge call attention to the fact that calcium oxalate crystals are present in the urine in over 40 per cent. of cases without jaundice, but in only 6 per cent. of jaundiced cases.

The most important characteristic about pancreatic calculus is that it is composed of material impenetrable to the X-rays and therefore can be demonstrated on an X-ray plate. If shadows are found by the X-ray, or if a stone is passed having the characteristics of pancreatic calculus, the diagnosis is simple. In other cases it is tentative and can be cleared up only by operation.

Diagnosis.—Diagnosis depends on X-ray examination. Gall-stones are not shown in a skiagram. The composition of pancreatic stones, if any are passed, is diagnostic, as they are composed of calcium salts without cholesterin or biliary coloring matter. Other than these two signs there is no method of arriving at a definite diagnosis. Lichtheim, in 1894, made a diagnosis of pancreatic calculi in a patient who suffered from epigastric colics, and later developed diabetes and characteristic diarrhœa; and autopsy confirmed the diagnosis. Pepper, as long ago as 1882, made the diagnosis of pancreatic calculus in a patient under his care, but this was not confirmed by the passage of calculi, nor did the patient come to autopsy. Kinnicutt collected seven cases in which the diagnosis was made during life, and Cipriani has reported another case apparently overlooked by Kinnicutt.

Treatment.—Operation was suggested by Körte in 1898; it is the only rational method of treatment. The stone cannot be absorbed and while one may be passed occasionally, usually there are more left. As the condition is so often associated with chronic pancreatitis the latter also calls for operation, at which time the stone can be removed. Very often operation is undertaken because of a mistaken diagnosis of cholelithiasis or cholecystitis.

The treatment of colic is symptomatic as in gall-stones.

The *operation* consists in cutting down through the substance of the gland and removing the stone wherever it may be. In operation on the head of the pancreas it may be necessary to

mobilize the duodenum to gain access to the affected area. In some cases the calculus or calculi may be exposed just to the left of the descending portion of the duodenum. If the stones are in the body or toward the tail of the pancreas, they are exposed best by division of the gastro-colic omentum. Cut surfaces of the pancreas unite after suture despite the presence of pancreatic juice on the surfaces of the wound; but in every case efficient drainage must be provided down to the site of the sutured area.

Fig. 26.—Pancreatostomy. (*Link.*)

Link has collected six operations for stone in the pancreas. In his own patient, constituting the seventh operation on record, he performed a formal *pancreatostomy*. Finding the pancreas even up to its tail filled with innumerable small stones, making it feel like a bag of fine sand, he exposed it through the transverse meso-colon. He next tore through the peritoneum covering the

pancreas, seized the tail of the pancreas, and commenced its enucleation just as if it were a pyosalpinx covered with adhesions. He found it a comparatively easy task to free the gland as far to the patient's right as the superior mesenteric artery. It was now possible to bring the tail of the pancreas out of the abdominal wound. Sponges were placed in its bed to arrest hemorrhage, which was not at all alarming; the pancreatic branches of the splenic artery were not ligated. After isolating the pancreas by gauze, it was split in the middle line for about two-thirds of its length. This opened the dilated duct of Wirsung, which was found to be filled with small facetted stones along its entire length. The stones, except those in the head of the gland, were removed; and a drainage tube was laid in the dilated duct of Wirsung, projecting several inches from the tail of the pancreas. The gland was then closed around the tube with a continuous suture of No. 1 chromic catgut (Fig. 26). After stitching the opening in the meso-colon to the body of the pancreas, the great omentum was sutured over the sutured area of the pancreas. A gauze drain was placed beneath the pancreas, to protect it from the small intestines. Finally the abdominal wound was closed around the tail of the pancreas, which emerged at the lower angle of the incision. Pancreatic fluid was discharged freely at first (about 24 ounces daily during the first week), but as the pancreatic inflammation subsided the secretions resumed their natural course into the duodenum. Several months after operation the drainage was slight, and the patient had resumed her usual active life.

REFERENCES.

Cipriani: Therap. Monatsh., 1898, xii, 617.
Johnston: Amer. Jour. Med. Sc., 1883, lxxxvi, 404.
Kinnicutt: Amer. Jour. Med. Sc., 1902, cxxiv, 948.
Körte: Die Chir. Krankh. u. d. Verletzungen d. Pankreas. Deutsche Chir., Lief. 45 d. Stuttgart, 1898, S. 206.
Lazarus: Beitr. z. Path. u. Ther. d. Pankreaserkrankungen, Berlin, 1904.
Lichtheim: Berl. klin. Woch., 1894, xxxi, 185.
Link: Annals of Surgery, 1911, i, 768.
Pepper: Med. News, 1882, ii, 705.

Injuries of the Pancreas.

Uncomplicated injuries of the pancreas are very rare. This is to be explained from its deep situation, almost completely covered by other abdominal organs. Körte showed that in twenty out of thirty cadavers the pancreas was entirely covered by neighboring viscera. On account of its inaccessibility, rupture is less seldom uncomplicated than either gunshot or stab wound. Föwelin refers to twenty-nine cases of *isolated rupture* of the pancreas. The patient under his own care was the only case he could find of *uncomplicated stab wound;* the instrument entered from behind, to the left of the spine. Becker appears to have reported the only *uncomplicated gunshot wound* of the pancreas on record; the bullet passed to the left of the stomach through the gastro-splenic omentum. Both Föwelin's and Becker's patients recovered after prompt operation.

The *symptoms* of pancreatic injury are in no way characteristic, and an accurate diagnosis before opening the abdomen usually is impossible. Usually it is the complicating injuries (liver, stomach, etc.) which produce recognizable symptoms, and the lesion of the pancreas is discovered only incidentally. Wohlgemuth and Benczur have pointed out that there is an increase in the amount of diastase in the blood and urine within a few hours after experimental or pathological obstruction of the pancreatic duct; and Noguchi suggests that chemical tests for diastase may be of value in determining whether or not the pancreas has been injured in cases of abdominal traumatism.

The *prognosis* depends upon the promptness of operation and very largely upon the presence of complicating injuries. Among twenty-nine cases of *isolated rupture* of the pancreas mentioned by Föwelin, seven died without operation; twenty-two were operated on, with fourteen recoveries and eight deaths, a mortality of 36.3 per cent. Diehl collected twenty-two cases of *gunshot wounds* of the pancreas, only one of which was uncomplicated: among these patients, six died without operation; sixteen were operated on, with nine recoveries and seven deaths a mortality of 43.7 per cent. *If the wound of the pancreas is not discovered at the time of operation, death nearly always is the result of overlooking it.* This was the cause of death in three out of the above seven fatal cases. If

operation is postponed, fat necrosis and retroperitoneal hemorrhages may be found when the abdomen is opened; the case then will closely resemble one of acute pancreatitis.

Treatment.—Immediate laparotomy is indicated as in all injuries of the abdominal viscera. Usually the incision is made in or near the median line, for purposes of exploration. In every case in which there is any possibility, even remote, of pancreatic injury, the pancreas should be explored, either through the gastro-colic or the gastro-hepatic omentum. The former is the preferable route, as giving better exposure. As directed in Vol.I, page 323, the incision in the gastro-colic omentum should be *at least three inches long*, and should pass below the gastro-epiploic vessels (Fig. 21). Bleeding from the pancreas should be checked by gauze packing, unless the point is freely accessible when suture may be attempted. But even where the injury has been securely sutured, the surgeon should not neglect to drain the injured region. Should a pancreatic fistula develop, the discharge may be much lessened and healing accelerated by putting the patient on Wohlgemuth's antidiabetic diet (page 311).

REFERENCES.

Becker: Beitr. z. klin. Chir., 1904, xliv, 748.
Benczur: Wien. klin. Woch., 1910, xxiii, 890.
Diehl: Beitr. z. klin. Chir., 1911, lxxiii, 206.
Föwelin: Arch f. klin. Chir., 1911, xcv, 1014.
Körte: Deutsche Chir., Lief. 45 d., Stuttgart, 1898.
Noguchi: Arch f. klin Chir., 1912, xcviii, 545.
Wohlgemuth: Berl. klin. Woch., 1910, xlvii, 92.

CHAPTER VII.

TUMORS OF THE PANCREAS.

Solid Tumors of the Pancreas.

These include **carcinoma, sarcoma** and **adenoma,** of which carcinoma is the most common.

Carcinoma of the Pancreas.—Various statistics have been reported indicating the frequency of carcinoma of the pancreas. But figures derived from records published before 1900 are not entirely trustworthy, as until the general recognition of chronic pancreatitis which followed Mayo Robson's researches, many cases of the latter disease were regarded as carcinomatous. Without histological examination it is difficult to distinguish one from the other. With these limitations in mind, the following classical statistics may be quoted: among 33,788 cases of carcinoma in males Bashford found *primary carcinoma* of the pancreas in 526; and among 50,660 cases in females, the tumor was *primary in the pancreas* in 474. This shows a distinctly greater frequency of primary carcinoma in the male sex.

Secondary carcinoma of the pancreas is of little interest to the surgeon. It seldom is the result of metastasis; in almost all cases it is due to extension from neighboring organs, particularly the stomach. Secondary carcinoma has been considered much more common than the primary form, only two of Eppinger's nineteen cases being primary. But Hale White's statistics do not uphold these figures, and Ferguson says that primary carcinoma of the pancreas is more common than secondary. If Oser's statement is true that 10 per cent. of all cases of primary carcinoma of the stomach involve the pancreas secondarily, there can be no doubt that secondary growths are more common than primary.

Age.—Carcinoma of the pancreas is a disease of middle or advanced life. In nearly all reported cases the patient has been over forty years of age. Cases occurring in childhood have been reported by Bohn, Kühn, Simon and Dutil. Their cases were

respectively seven months, two years, thirteen years and fourteen years of age.

Sex.—As indicated already, carcinoma of the pancreas is more common in men than women, the relation being about three to two, much the same ratio which obtains in cases of chronic pancreatitis. Mirallié reported 106 cases of primary pancreatic cancer, sixty-nine in men and thirty-seven in women. Oser, including Mirallié's cases, reported 246 in men to 142 in women. Among the sixteen cases of primary carcinoma arising in the body of the pancreas, studied by Leriche, eleven occurred in males, and only five in females.

Pathology.—While diffuse growths occasionally occur, primary carcinoma usually is of small size. This fact explains why primary involvement of the head of the pancreas with metastasis to the liver is so often mistaken for primary carcinoma of the liver, the original growth in the pancreas being overlooked on account of its small bulk. When a pancreatic cancer grows to a large size, invades neighboring structures and forms extensive adhesions, the starting point of the growth is not easily determined.

In primary carcinoma the head of the gland is the most frequent site of the growth, although the body, tail or whole gland may be involved. Oser in sixty-eight cases found the following distribution: head thirty-nine; whole organ nineteen; tail four; head and body three; body and tail one; head and tail one. In Mirallié's 106 cases the head was the site of the growth in eighty-two. In Segré's cases the distribution was as follows: head, thirty-five; entire gland, nineteen; body, two; tail, one. Robson and Cammidge give the following percentages: head 62 per cent.; tail 5.5%; body 3.5 per cent.; diffuse growth 29 per cent.

The point of origin of the growth usually determines its type (Hulst). If it arises in the epithelium of the excretory ducts it develops into a cylindrical celled tumor of the adenocarcinomatous form. Letulle contends that a carcinoma springing from the duct of Wirsung is composed of solid alveoli of sphercidal and not cylindrical cells. Olivier reported a case in which the cells of the tumor apparently arose directly from the ducts and the growth was of the spheroidal-celled solid alveolar type without the central lumen seen in adenocarcinoma. Hulst thinks

that these spheroidal-celled tumors with solid alveoli take their origin from the glandular epithelium.

Whatever the origin of these two forms there usually is developed sufficient fibrous tissue to make them hard and firm. The great majority of pancreatic carcinomata are of the *scirrhous variety*. Occasionally the growths are very cellular, forming a tumor of *encephaloid type*. Columnar-celled tumors may undergo *colloid degeneration*, several having been reported.

No connection has ever been definitely determined between the islands of Langerhans and the origin of carcinoma. Hillier and Goodall describe a form of carcinoma supposed to arise from the islands of Langerhans. These differ from the other forms in the great irregularity of their cells. Olivier believes that some of the so-called cases of primary carcinoma of the pancreas arise in the duodenum from the glands of Brunner.

Sooner or later the growth of a carcinoma of the pancreas encroaches on one of the excretory ducts and causes complete obliteration of its lumen. This results in the development of a chronic interstitial pancreatitis in the area drained by the affected duct. Moreover, the tumor itself, as already noted, is surrounded by a reactionary growth of connective tissue.

Carcinomatous degeneration in chronic interstitial pancreatitis is spoken of as a possible complication by Hulst, but has not yet been proved to occur.

Neighboring organs may be involved from primary carcinoma of the pancreas by metastasis, extension, or the formation of adhesions.

Metastasis occurs in the liver as a rule. When it is extensive the determination of the site of the primary growth is extremely difficult. This applies particularly to cases of carcinomatosis said to arise from primary tumors of the pancreas.

The position, size, and direction of the growth of the tumor determine the organs involved by extension or adhesions. Carcinoma of the head of the pancreas encroaches on the common bile-duct, and causes steadily increasing obstruction until the lumen is completely obliterated. This blocking of the common duct is evidenced by progressively deepening jaundice, which usually is associated with enlargement of the gall-bladder, unless previous gall-stone disease has caused thickening and shrinkage from fibrous

change. The stomach and duodenum from their relations to the pancreas are naturally the areas to suffer most frequently. Obstruction to the duodenum or pylorus causing dilatation of the stomach, ulceration into the duodenum or stomach, compression of the transverse colon or stomach and of various large vessels in the abdominal cavity, and even compression of the left ureter causing hydronephrosis, have all been described as resulting from carcinoma of the pancreas.

Glycosuria is not a frequent complication but when it does occur it indicates involvement of the islands of Langerhans, either in the chronic pancreatitis resulting from the growth or in the tumor itself.

Symptoms.—In carcinoma of the pancreas there are no pathognomonic symptoms. Following the original clinical picture drawn by Bard and Pic, it was considered for a long time that steadily increasing jaundice, enlargement of the gall-bladder and rapid emaciation constituted a characteristic syndrome or group of symptoms, which indicated carcinoma of the head of the pancreas. Now, however, it is generally recognized that this syndrome may result from any condition which causes obstruction at the papilla of Vater. In carcinoma of the pancreas it is only when the growth is in the head of the gland that this obstruction results, so there are necessarily cases that do not exhibit the so-called characteristic symptoms. On the other hand various conditions, such as carcinoma of the bile-ducts, chronic interstitial pancreatitis, carcinoma of the gall-bladder, etc., may cause obstruction of the choledochus and of the duct of Wirsung, and thus give rise to this same group of symptoms. A diagnosis of carcinoma of the pancreas, therefore, must be made with a certain degree of reserve; and only after careful consideration of the history, physical signs and laboratory findings in each individual case.

Jaundice.—In carcinoma of the head of the pancreas the common duct is steadily compressed and eventually occluded by the growth, jaundice resulting. The characteristic feature of the jaundice is steady progress without intermission or remission, differing in this particular from that caused by a stone in the duct, which is subject to variation, as obstruction is seldom so complete as to prevent absolutely the passage of bile. Complete obstruction is shown by the absence of stercobilin in the fæces. Occlu-

sion of the common duct by cancer of the choledochus, or of the papilla of Vater, or by extension from cancer of the gall-bladder or pylorus gives rise to the same type of jaundice, and it is indistinguishable from that caused by carcinoma of the head of the pancreas.

The manufacture of bile continues in spite of the obstruction and the jaundice gradually deepens until it becomes the "black jaundice" of older writers.

If the body or tail of the pancreas is affected the disease may run its whole course without causing jaundice. As a symptom of carcinoma of the pancreas, jaundice is peculiar to growths involving the head of the gland, the only position in which the tumor can compress the common duct.

Extension to the head of the pancreas or metastasis to the liver or the lymph-nodes in the gastro-hepatic omentum are necessary before jaundice will arise in cases of carcinoma of the body or tail of the pancreas.

Enlargement of the Gall-bladder.—If carcinoma is the primary condition and there has been no previous gall-stone disease or duct inflammation; in other words, if at the time of onset of obstructive jaundice the gall-bladder is normal, back pressure on the ducts causes distention of the gall-bladder from the accumulation of bile. As the obstruction increases and the pressure becomes greater, no more bile can reach the gall-bladder and the continuing distention is due to secretion of mucus.

If there has been previous inflammation from stones or noncalculous infection the deposit of fibrous tissue in and around the walls of the gall-bladder interferes with its elasticity and it is consequently incapable of distention. In these cases the gall-bladder is small and contracted irrespective of the amount of back pressure.

Pain.—Opinions differ in regard to the occurrence and the degree of pain. Opie says, "Pain is one of the earliest and most common symptoms." On the other hand Robson and Cammidge say, "Pain is usually absent or unimportant"; while Robson and Moynihan say, "In one-half the cases at least, the suffering is exquisite." Chauffard called particular attention to very severe crises of abdominal pain, which he described as a veritable visceralgia. He described the patients as sitting with flaccid ab-

domen, bending forward to their knees, and unable to eat anything. From these contradictory statements it may be inferred that all varieties in character and severity of pain occur; and that, while pain may be a conspicuous feature, it is not a necessary factor in the clinical picture of a case of pancreatic carcinoma.

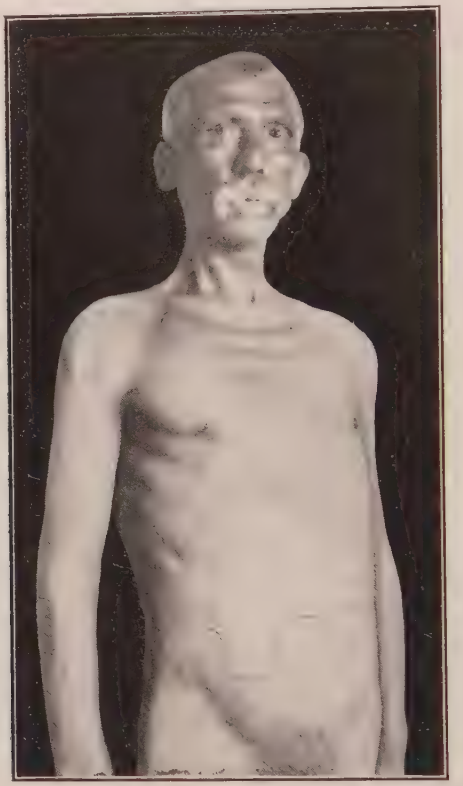

Fig. 27.—Obstructive Jaundice with Enlargement of the Liver and Gall-bladder, Presumably from Carcinoma of the Head of the Pancreas. An Inoperable Case. (*From a patient in the German Hospital.*)

Various explanations of the cause have been given. Continuous pain increasing in severity until death is ascribed to pressure on the cœliac ganglia. Colicky pain is supposedly due to obstruction of the duct of Wirsung or the common duct setting up attacks of true colic. When the head of the pancreas is involved, the usual seat of pain is in the epigastrium or right hypochondrium, from which it may radiate to the back or shoulder. When the

growth originates in the body of the gland, Leriche says the earliest pain is in the left hypochondrium.

Loss of Weight and Strength.—Early in the course of the disease the loss of weight and strength is very striking, and this continues progressively and rapidly until the patient dies from exhaustion. Death seldom is delayed more than a few months after the appearance of jaundice. Pancreatic cancer is said to be the most rapidly fatal of all forms of malignant disease. The entire duration of the malady, from the recognition of the first symptoms, seldom exceeds a year.

Digestive Disturbances.—These are of two kinds, (1) those due to obstruction of the duodenum or pylorus, causing symptoms of stricture, and (2) those due to interference with pancreatic digestion. The former give the usual picture of pyloric obstruction, anorexia, distress and distention after eating, and delayed vomiting, together with the well-known physical signs and laboratory findings of gastric dilatation from obstruction.

If the duct of Wirsung is occluded to such an extent that little or no pancreatic juice reaches the intestine, the usual evidences of *pancreatic insufficiency* are found. There is early loss of weight and strength, much greater than that usually caused by the growth of a malignant tumor.

The changes that occur in the fæces when pancreatic juice does not reach the intestine have been fully described at page 274. Briefly, they are increase in the amount of unabsorbed fat in the fæces from the normal of 5 per cent. to 50 per cent. or more (in cancer of the head of the pancreas the unabsorbed fat in the fæces may amount to 90 per cent. of that ingested); diminution in the percentage of split fat from the normal of 70–80 per cent. to as low as 15 per cent. Even in those cases in which there is no increase above normal in the actual amount of fæcal fat, there is still a diminution in the proportion of split fats to 40 per cent. or less. These findings and their significance have been discussed at some length in relation to diagnosis (page 335).

Tumor.—Tenderness is unusual, but may occasionally be present and interfere with palpation. In the majority of cases no mass referable to the pancreas is palpable, the tumor usually being small and confined to the head of the gland. While it is true that the greater number of malignant growths of the pancreas

are fixed, this is not a universal rule by any means. Although the pancreas is retroperitoneal, it is invested by loose areolar tissue which permits considerable motion, and unless it is anchored by inflammatory or malignant adhesions, mobility is marked. Mobility of a tumor, therefore, does not necessarily rule out a pancreatic origin. Body and tail tumors are more likely to be movable than those arising in the head of the gland. As a rule, however, malignant growths are either too small to be felt or are firmly fixed by adhesions.

In thin patients with relaxed abdominal walls any considerable enlargement of the pancreas can be detected, but it takes a comparatively large tumor to be recognized as such. When palpable it usually transmits pulsation from the aorta; and if, as in Poncet's case, there is also a murmur the lesion may be mistaken for an aortic aneurysm. Mirallié reported that there was a palpable tumor in one-fourth to one-fifth of his 113 cases. A tumor was palpable in nine out of fourteen cases of carcinoma of the body or tail, analyzed by Leriche; usually the mass presented through the gastro-hepatic omentum, displacing the stomach downward. Although the pancreas itself usually cannot be palpated there often is an epigastric tumor due to the distended gall-bladder. In rare instances two tumors have been felt, one the pancreatic growth and the other the distended gall-bladder. An enlarged gall-bladder is easily felt as a rounded, smooth tumor which is just beneath the abdominal wall and moves with respiration. In sixty-two cases of obstruction of the common bile-duct due to carcinoma of the pancreas reported by Ecklin, in 58 there was recognizable enlargement of the gall-bladder.

The liver has no distinctive features, its size varying from normal to marked enlargement, depending upon the degree and character of the secondary involvement. Considerable increase in the size of the liver is to be expected in the late stages of the disease (Fig. 27).

Glycosuria occurs in a certain proportion of cases. It appears when the growth has advanced to such an extent that it has destroyed the greater number of the islands of Langerhans; as in the case of chronic interlobular pancreatitis, the islands of Langerhans persist for some time after the parenchyma has been destroyed, so that the appearance of sugar in the urine usually is

a late development, if it occurs at all. Mirallié reported glycosuria present in thirteen of fifty cases. Both alimentary glycosuria and transient glycosuria have been reported, but the mere presence of sugar in the urine does not serve to differentiate the form of pancreatic disease causing it.

In cases of continued jaundice there always is a *tendency to hemorrhage*. In cancer of the head of the pancreas, gastric, intestinal, subcutaneous, and even nasal and oral hemorrhages are not uncommon. Spontaneous hemorrhage usually is not serious, but that which occurs after operation is distinctly dangerous. Persistent and uncontrollable postoperative oozing frequently is fatal even in those cases where nothing but an exploratory incision has been undertaken. This, too, in spite of the fact that calcium has been given freely before operation.

The *Cammidge reaction* is said to be present only when the growth of the carcinoma gives rise to a chronic interstitial pancreatitis. It was present in four out of eight of our cases of carcinoma of the pancreas. Robson and Cammidge report four positive reactions in sixteen cases.

The analysis of *stomach contents* is comparatively of little value in differentiating pancreatic from other upper abdominal growths, but in doubtful cases useful confirmatory evidence is occasionally obtained.

The *absence of stercobilin in the fæces* is a valuable sign in that it signifies complete obstruction to the flow of bile, thus indicating that the common duct is *absolutely occluded*. In gallstones, pancreatitis, cancer of the liver and simple cholangeitis, stercobilin never is constantly absent from the fæces; this fact indicating that obstruction is not complete. Cancer of the head of the pancreas and that of the common duct are the two conditions that completely obstruct the outflow of bile.

Left-sided hydronephrosis, ascites, œdema of the legs and enlargement of the spleen sometimes are present as pressure symptoms. Another pressure symptom has been mentioned at page 360, this is aortic pulsation with a murmur. Chylous ascites has been reported; it is due to rupture of the thoracic duct.

Elevation of temperature results only when an intercurrent infection arises.

The **clinical course** of a case of carcinoma of the pancreas

is remarkable for its rapid progress to a fatal termination. The interference with pancreatic digestion, the jaundice, and the cachexia from a malignant tumor combine to make this one of the most rapidly fatal of malignant diseases. Death usually ensues within six to eight months after the appearance of jaundice (Robson and Cammidge), but it may be delayed for two or even four years after the onset of the earliest symptoms (Opie).

Diagnosis.—In a typical case of primary carcinoma of the head of the pancreas the diagnosis can be made with a fair degree of certainty, but unfortunately not at an early period of the malady unless the growth arises very close to the main excretory duct and rapidly invades the papilla of Vater or compresses the common bile-duct. The patient, usually over forty years old, complains for an indefinite period of indefinite upper abdominal symptoms having no localizing character. After a longer or shorter time jaundice appears and painlessly and continuously deepens; the gall-bladder enlarges and the patient loses weight and strength very rapidly. Pain may or may not be a conspicuous feature. The persistent absence of stercobilin in the fæces is a valuable confirmatory sign, and indicates complete absence of bile from the intestine. The fæces may show signs characteristic of failure of pancreatic digestion due to the absence of pancreatic juice. If interstitial pancreatitis is associated with the cancer, Cammidge's reaction probably will be positive. It is positive in 25 per cent. of cases. A tumor connected with the pancreas seldom is recognizable. When there is such a tumor its relations may be determined by inflating the stomach and colon. Theoretically this should completely obscure a tumor of the pancreas, but from the number of cases reported where operation was undertaken for other conditions, it is evident that pancreatic tumors often exhibit characteristics common to various abdominal tumors. The size of the liver is of comparatively little value in differential diagnosis.

The main factors on which a diagnosis of cancer of the head of the pancreas is based are progressive jaundice, enlargement of the gall-bladder, rapid loss of weight and strength, and absence of stercobilin in the fæces. Such a clinical picture indicates complete obstruction of the common duct, and carcinoma of the head of the pancreas is the commonest cause of such obstruction,

yet other causes cannot always be excluded, especially carcinoma of the common bile-duct or the papilla of Vater causing coincident obstruction of the duct of Wirsung. Carcinoma of the hepaticus causes obstructive jaundice but there is no pancreatic insufficiency; and even when the duct of Wirsung is obstructed by a growth, pancreatic insufficiency may not develop if the accessory duct of Santorini is patulous.

Atypical cases of carcinoma of the pancreas which develop after symptoms of gall-stones, gastric or duodenal disease, as well as those in which obstruction of the choledochus does not occur, and those exhibiting a palpable, movable tumor, cause extreme difficulty in diagnosis and it is at times impossible to make a positive diagnosis.

Carcinoma in the body or tail of the pancreas is practically incapable of differentiation from other palpable tumors in the upper abdomen, for it may be as freely movable as any of the commoner tumors, and will not cause obstructive jaundice nor symptoms of pancreatic insufficiency. Gastric and intestinal symptoms and the more superficial position of the tumor favor the diagnosis of an extra-pancreatic origin, but much reliance cannot be placed on these points.

Differential Diagnosis. *Carcinoma of the Common Duct.*—For practical purposes this includes carcinoma of the papilla, of the ampulla of Vater, of the common bile-duct and of the common hepatic ducts.

Primary carcinoma of the common bile-duct is very much less frequent than carcinoma of the pancreas. Contrary to what might be expected, gall-stone disease is not a predisposing factor in cancer of the bile-passages.

In primary carcinoma of the common duct the onset usually is insidious and after a varying period in which ill-defined epigastric symptoms are mainfested, jaundice appears and deepens gradually but persistently. So far the resemblance to pancreatic carcinoma is striking, but until the pancreatic ducts are obstructed by the growth in the choledochus there are no signs of pancreatic insufficiency. From this time on the case runs the same clinical course as carcinoma of the head of the pancreas and the two conditions are, as a rule, incapable of differentiation, except on the grounds that carcinoma of the head of the pancreas is much

commoner than common duct cancer and is associated with more rapid emaciation and loss of strength because of the early interference with pancreatic digestion.

Carcinoma of the gall-bladder simulates cancer of the head of the pancreas only when the growth has extended to and occluded the ampulla of Vater, causing both obstructive jaundice and pancreatic insufficiency. When jaundice is due to involvement of the liver, and not to obstruction of the choledochus, stercobilin is not absent from the fæces.

The history of gall-stone disease or of prolonged upper abdominal symptoms, the comparatively late onset of jaundice, the absence of the smooth, rounded, gall-bladder tumor and in its place a hard, nodular tumor, or no tumor at all, make a clinical picture much different from that of carcinoma of the head of the pancreas.

Extension of malignant disease to the ducts from neighboring organs is preceded by symptoms of the primary disease, and jaundice is a late symptom.

Stone in the Common Duct.—The history of previous attacks, the onset of jaundice after colic, the intermittent character of the jaundice, the fact that there is little or no loss of weight and strength for a long time after the initial symptoms, the absence of a gall-bladder tumor and the presence of stercobilin in the fæces, all serve to differentiate gall-stones from pancreatic cancer. In addition, obstruction of the common duct by a stone usually gives rise to intermittent fever, while the temperature is normal or subnormal in cancer of the pancreas. Rigidity of the right upper rectus is also a frequent accompaniment of cholelithiasis.

Chronic Interstitial Pancreatitis.—When the abdomen is opened and the pancreas examined directly by sight and touch it often is impossible to differentiate carcinoma and pancreatitis. The presence or absence of Cammidge's reaction, of stercobilin in the fæces, a preceding history of gall-stone disease or other predisposing factor to pancreatitis, and, as a rule, the slower loss of weight and strength in pancreatitis help in differentiation. In inflammatory disease the gall-bladder usually is contracted; but even when it is distended it is sometimes possible to distinguish it from the distended gall-bladder which results from malignant obstruction by its friablity. In malignant disease the gall-blad-

der, unless itself involved in the carcinomatous process, is much more resistant and sutures inserted in it are much less apt to tear out than in the case of a gall-bladder which is the seat of long-standing inflammatory change.

In undetermined cases the patient should be given the benefit of the doubt and treated as for pancreatitis.

Cancer of the Liver.—Absence of the evidence of complete obstruction of the bile-ducts, the late onset of jaundice, enlargement of the liver, palpable nodules on the surface of the liver, and lack of symptoms and signs due to interference with pancreatic digestion indicate the probable diagnosis.

Cancer of the Pylorus.—Even if jaundice does occur it is a late symptom due to extension or metastasis. The gastric symptoms predominate although pyloric and pancreatic cancer may occur apparently at the same time and the primary seat of the tumor be uncertain.

The *gastric crises of tabes* may be distinguished from the visceralgia which characterizes some cases of pancreatic carcinoma by attention to other signs of locomotor ataxia.

Treatment.—Medical treatment is symptomatic.

In cases of doubt abdominal section is indicated as the condition may be amenable to operative treatment. Confluent enlarged lymph-nodes point to carcinoma while discrete glandular enlargement usually indicates pancreatitis. In nearly every case of carcinoma of the pancreas the disease has progressed too far to permit removal. Complete removal of the pancreas appears to be necessarily fatal. The case attributed to Billroth (1884) of recovery after complete extirpation of the pancreas, is rejected by Sauvé, who says it is cited everywhere but nowhere reported. In cancer of the body and tail a number of successful removals have been recorded. In 1910 Finney collected six resections of the body or tail of the pancreas with two deaths from the operation, two survivals for a few months, one reported as "recovered" but with no after history, and Finney's own patient (cystadenoma) who was in good health sixteen months after operation. In excisions of the tail, the stump may be closed, and any fistula may be expected to close under antidiabetic diet. In resections of the body, the head and tail should be sutured together, as in Finney's own case.

Compared to the number of actual cases, successful removal must be looked upon as only rarely possible, but should always be undertaken if there is the slightest possible chance of success. Sauvé quotes Terrier's maxim: *Dans le doute, ne t'abstiens jamais.*

A *radical operation* for carcinoma of the *head of the pancreas* involves also removal of the second or descending portion of the duodenum. The operative technique has been well systematized by Desjardins and by Sauvé, the latter of whom uses the term cephalic duodeno-pancreatectomy to describe the operation. He collected sixteen cases of cephalic pancreatectomy of which the details are known, and refers to five others merely mentioned in journals. Of these sixteen patients nine survived the operation, (Ruggi, Sendler, Codivilla, Biondi, Tricomi, Franke, Duval, Villaréal, Mauclaire) and three patients were in good health a year later, but *these three did not have cancer* (Sendler, Biondi, Duval). Of all the operations collected by Sauvé that of Codivilla was the most ideally complete: cephalic duodeno-pancreatectomy, with gastro-jejunostomy in-Y, and cholecystenterostomy; his patient lived twenty-four days, and succumbed then to the preexistent cachexia. The patients of Tricomi and Franke survived for five months and six months, respectively. Cordoy, Mauclaire, Villaréal, and Moynihan employed duodeno-pancreatectomy; while Cunéo adopted the method employed by Tuffier in a case of carcinoma of the papilla of Vater invading the pancreas. This consisted in enucleation of the head of the pancreas and excision of the ampulla of Vater; the stump of the pancreas was fixed in the abdominal wound. Even in the technique recommended by Sauvé, and which is described in detail at page 477, the stump of the pancreas is fixed in the abdominal wound. In the technique proposed by Desjardins the pancreatic stump is implanted into the intestine. Coffey, in experimental work on dogs, sought a technique to facilitate such a step. The best way to do this, he found, was to throw the lumen of two intestines into one by using a loop, and thus allow ample room for invagination of the pancreas (page 480).

Cholecystostomy and cholecystenterostomy do not prolong life to any extent and a large proportion of the patients die as a result of the operation. The results of this operation in cases of

malignant disease have been noted at pages 131 and 226. From these statistics it is evident that operation is of little or no value in cases of carcinoma of the head of the pancreas either in effecting a cure or prolonging life. In cancer of the body or tail the prospect is slightly better but not at all hopeful.

Sarcoma of the Pancreas.—Kakels in 1902 collected twenty-one cases. Primary sarcoma is very rare and while secondary involvement occurs more frequently it is still an uncommon condition. In 1909 Ravenna recorded from the literature twenty cases of primary sarcoma and reported two original cases. Villard and Stéfani have since recorded an operation in which marsupialization was done for polycystic sarcoma of the pancreas; death occurred eight days later.

Primary sarcoma is of the small round-celled variety, or lymphosarcoma. *Secondary* sarcoma is also usually lymphosarcoma and arises from the mediastinal or abdominal lymph-nodes or from the duodenum. A number of cases of melanotic sarcoma are reported and usually have resulted from metastasis from the eye.

Segré reported two primary sarcomas occurring in 11,492 autopsies. Hale White found one in 6708 autopsies. In Finney's report of seventeen cases of resection of the pancreas, sarcoma occurred four times, two of the patients recovering. One of these died of recurrence within a few months.

Diagnosis is of course not possible before operation.

If the tumor is in the body or tail successful removal is possible. If the growth is in the head of the gland cephalic duodeno-pancreatectomy may be done but usually patients are seen too late for radical operation to be successful.

Adenoma is of pathological rather than surgical interest (Cecil). The diagnosis is made by microscopic examination and there are no characteristic symptoms or physical signs. A certain number of successful removals have been reported.

REFERENCES.

Bard and Pic: Revue de Méd., 1888, viii, 257; 363.
Cecil: Proc. New York Path. Soc., 1910–11, n. s., x, 135.
Cecil: Jour. Exper. Med., 1911, xiii, 595; xiv, 500.
Chauffard: Bull. de l' Académie de Méd., Paris, 1908, lx, 242.

Coffey: Annals of Surg., 1909, ii, 1238.
Desjardins: Rev. de Chir., 1907, xxxv, 945.
Ecklin: Diss. Inaug., Basel, 1896.[1]
Ferguson: Surg., Gyn. and Obst. 1910, x, 393.
Finney: Annals of Surg., 1910, li, 818.
Hillier and Goodall: Archives of Middlesex Hospital, London, 1904, ii, 1.
Hulst: Virchow's Archiv. f. path. Anat., 1905, clxxx, 288.
Kakels: Amer. Jour. Med. Sc., 1902, cxxiii, 471.
Leriche: Arch. f. klin. Chir., 1910, xcii, 1048.
Mirallié: Gas. des Hôpitaux, 1893, lxvi, 889.
Olivier: Étude sur le developpement du Cancer Pancréatique. (Lausanne). Jena, 1894.[1]
Oser: Diseases of the Pancreas. Nothnagel's Encyclopædia; English Translation, Philadelphia, 1903, p. 17.
Poncet: 1890, cited by Leriche, loc. cit.
Ravenna: Il Policlinico, 1909, xvi, 166, Sez. Chir.
Sauvé: Rev. de Chir., 1908, xxxvii, 113; 335.
Segré: Ann. Universal. della Med. e. d. Chir., 1888, cclxxxiii, 3.
White Hale: Guy's Hospital Reports, 1897, liv, 17.

Cysts of the Pancreas.

The percentage incidence of pancreatic cysts is difficult to determine because the majority of collected cases are reports of operation, most often only incision and drainage, where the true origin and connections of the tumor could not be ascertained with any degree of accuracy. For this reason it is probable that many cases are classed as pancreatic although the cysts have arisen independently of the pancreas.

Classification.—The simplest and most convenient classification is into true cysts and false or pseudocysts. The former include those due to retention of pancreatic secretion, cystic new growths, hydatid cysts and congenital cystic disease. Pseudocysts arise in close association with the pancreas and involve it secondarily. These cysts usually are formed by effusions the result of abdominal injuries. Some authors classify with these extra-pancreatic pseudocysts those which arise in the pancreas as a result of hemorrhage from injury or acute pancreatitis. As these are practically indistinguishable from true retention cysts such a classification is not rational from a clinical standpoint.

Ætiology. *Age.*—The years between twenty and forty fur-

[1] These works have not been accessible to us.

nish most of the cases. Railton reported a pancreatic cyst in a six months old child; Shattuck, one in a child thirteen months old; Connelly and Richardson, each one in a child fourteen months old; while Stieda reported a case in a man seventy-six years old.

Sex.—Robson and Cammidge say that true cysts are more common in women and pseudocysts more common in men, presumably because the latter are more exposed to injury. Körte's statistics show almost equal incidence in men and women.

Traumatism.—Experimental evidence shows that injury to the pancreas causing hæmatoma results in cyst formation. Lazarus reports eight cases of pancreatic cyst arising apparently as a result of traumatism. Thirty-three (28 per cent.) of the 117 cases collected by Körte were preceded by abdominal injury, these injuries usually being direct blows of varying degrees of violence; although falls, compression of the abdomen, etc., have also preceded and apparently caused a certain number of pancreatic cysts.

Previous Pathologicat Conditions.—All conditions likely to be associated with chronic interstitial pancreatitis have been described in connection with pancreatic cysts. Calculi, tumors, chronic infection of the duodenum and bile-passages, duodenal and gastric ulcer and pancreatic lymphangeitis have been mentioned as pathological lesions accompanying pancreatic cysts. Whether they cause both the cyst and the pancreatitis or whether the cysts result from the pancreatitis is hard to determine. Robson and Cammidge favor the latter supposition and think that when cyst, calculus and chronic pancreatitis occur together the two former have a common cause in the latter.

Acute hemorrhagic pancreatitis has frequently resulted in cyst formation.

Pathology. *Retention Cysts.*—It is doubtful if simple obstruction of the pancreatic duct or one of its branches leads to the formation of a cyst or cysts. Those observers who think that it does believe that there is a change in the character of the pancreatic juice, which as a result of stagnation becomes less easily absorbed. Subsequently this causes disintegration of the glandular tissue with the formation of a cavity into which secretion and exudate are poured out. Failure to cause cyst formation in certain cases is ascribed to non-closure of the duct of Santorini. After causing

only slight dilatation of the duct Senn suggested that complete obstruction would lead to atrophy, but that intermittent obstruction, as by a calculus, would cause cyst formation in the same manner that a stone in the ureter causes hydronephrosis. While such theories may furnish a partial explanation of the mode of origin of cysts, there are other unknown factors, as is shown by the fact that the same ætiological factors blamed for the formation of cysts are brought forward as direct causes of chronic interstitial pancreatitis. We have no knowledge as to why the same conditions at one time cause cyst formation and at another do not. There is always a certain amount of pancreatitis in association with cysts of the pancreas, and Robson and Cammidge express the opinion that the cyst is the direct result of the compression of the duct by contracting bands of scar tissue. This removes the difficulty only a step farther, as it does not explain why a cyst occurs in one case of pancreatitis and not in another.

Thiroloix altered the contents of the ducts before ligating both, by injecting soot and carbolized vaseline, and caused the formation of a cyst with, in addition, very advanced chronic pancreatitis. The determining factor of cyst formation in this case is supposed to have been the alteration of the pancreatic juice by the foreign matter injected, which interfered with absorption.

It is not yet clear that the chronic pancreatitis may not be the result of extension of the chronic inflammation always found around the wall of a cyst.

Pancreatic calculi, gall-stones, duodenal ulcer, tumors and lymphangeitis have all been found in association with retention cysts but their connection therewith is not understood. If they obstruct the duct this may cause a rapid increase in size of a preformed cyst, but there is no definite reason for saying that they are causative factors.

All sizes and conditions of cysts occur; uni- and multi-locular cysts, single and multiple, large and small, occur in all sorts of combinations and the ætiology of the various combinations is uncertain.

Virchow described a multiple dilatation of the duct of Wirsung, caused by obstruction from a tumor of the duodenum, which he termed "*ranula pancreatica*." Multiple small cysts, the result of obstruction of the smaller ducts, were named "*acne pancreatica*"

by Klebs. These two forms are uncommon and of no significance from the standpoint of diagnosis and surgical treatment.

Small cysts are recognized either at autopsy or in the course of upper abdominal operations for other conditions.

Large single or multi-locular cysts offer the best opportunity for diagnosis and treatment. They are said to be associated with obstruction of the large branches of the main duct and vary in size up to enormous proportions, cysts containing fifteen liters and more having been reported.

The walls of a retention cyst consist of dense fibrous tissue of varying thickness. The inner surface may be lined more or less completely by a single layer of cylindrical epithelium, indicating the origin of the cyst from a dilated duct. The absence of this epithelial layer is no proof that it is not a true retention cyst as the action of the cyst contents may destroy the epithelium. Portions of pancreatic tissue may be found included in the walls of these cysts, indicating a true pancreatic origin. Continued increase in size of a cyst causes more or less destruction of pancreatic tissue even in those cases where the growth is mostly away from the pancreas. The outer surface of the larger cysts is traversed by greatly distended blood-vessels.

Proliferation Cysts.—These cysts are formed by proliferation of the duct epithelium and accumulation of fluid. They are of two kinds, benign and malignant. Benign cysts have much the same characteristics as multi-locular ovarian cystadenomas. They are multi-locular, with small cysts in the walls of the larger ones, and frequently there are papilliferous growths on the inner surface. As a rule such a cyst is lined with columnar epithelium, but this may have been destroyed by the cyst contents.

A few cases of malignant cyst have been reported. They occur as epitheliomas without regular form and are usually made up of numbers of small cysts.

Remnants of the Wolffian body are said to give rise to tumors resembling proliferation cysts of the pancreas. All of these growths are rare.

Traumatic Cysts.—Differentiation of these cysts depends more upon the ætiological factor of injury than on any special pathological characteristics. Trauma may be a factor in causing either true or false cysts. The presence of blood in the cyst contents is

not demonstrative of traumatism, since hemorrhage may occur as the result of rupture of vessels in the wall of a cyst already formed; on the other hand, a cyst undoubtedly due to injury may contain clear watery fluid with no macroscopic evidence of blood. Occasionally a cyst has old clots adherent to the wall, the result of previous hemorrhage.

The fact that traumatism will give rise to pancreatic cysts receives ample confirmation from both experimental and clinical observations. Lazarus produced a cyst by crushing the pancreas of a dog, forming a hæmatoma, which later became an encapsulated cyst containing 100 c.c. of watery fluid.

Traumatic cysts are very frequently situated in the lesser peritoneal cavity, having little or no direct association with the pancreas.

Hydatid Cysts.—Hydatid cysts of the pancreas are extremely rare and present practically no symptoms or physical signs by which they can be recognized definitely as hydatid cysts of the pancreas in contradistinction to other cysts of the pancreas or hydatid cysts of neighboring viscera.

Congenital cystic disease is rare and has little or no clinical significance; it is evidenced by multiple small cysts as in congenital cystic disease of other organs. The cases of cyst reported in children from six to fourteen months of age probably are not cases of congenital cystic disease, as they had all the characteristics of retention cysts.

Contents.—The appearance and character of the fluid contents of pancreatic cysts and pseudocysts vary considerably.

The contents may be almost any *color*, but usually are light brown, although in many reported cases the cysts have contained clear watery fluid. The presence of blood in varying quantities influences the color, according to the length of time that has elapsed since the blood escaped. The presence of enzymes also causes alteration in the color and may be responsible for the loss of the products of hemorrhage in the cyst contents. Enzymes may also be responsible for the presence of blood in the fluid by causing erosion of the vessels in the wall of the cyst.

The *character* of the contents varies from watery fluid to a thick syrupy or colloid substance too thick to run freely through an aspirating trochar. It is purulent if suppuration has occurred.

Macroscopic examination therefore offers very slight assistance in determining the origin of such a cyst. Opinions differ as to the value to be attached to the chemical analysis and microscopic examination.

Chemical Analysis.—The presence in the cyst contents of various ferments, rich in the usual digestive powers of the pancreas, is strong presumptive evidence that the cyst is of pancreatic origin. This is undoubtedly true if the fluid digests albumen, starch and fat. When all three ferments are present it is practically certain that the cyst communicates directly with the pancreas, but the presence of only one (unless it is the fat-splitting ferment) is not diagnostic. Many cysts of undoubted pancreatic origin do not contain all three ferments and each one of the three has been demonstrated in cysts of extrapancreatic origin. The absence of pancreatic ferments in a true cyst is due to the fact that chronic disease of the pancreas interferes with its secretory function. Their presence in pseudocysts is due to communication with the parenchyma of the pancreas.

The other characteristics of pancreatic cyst fluids are: an alkaline reaction; a specific gravity from 1010 to 1020, although it may be much higher; the constant presence of albumen; the frequent presence of cholesterin; the occasional presence of mucin; and rarely the presence of traces of uræa.

Microscopic examination reveals red and white blood cells, epithelium, fat globules, necrotic tissue and frequently cholesterin crystals.

Symptoms and Physical Signs.—There are many recorded cases in which the only recorded symptom is the appearance and gradual growth of an abdominal tumor, but these cases are exceptional. The presence of the tumor and the associated inflammatory and pressure changes usually result in marked digestive and constitutional changes. Pain also is a fairly constant symptom.

Pain.—This varies in severity from a feeling of discomfort or distention to attacks of severe lancinating pain common to the serious abdominal crises. Pain may be continuous, intermittent, or continuous with acute exacerbations. At times the pain is severe enough to resemble acute intestinal obstruction, and often is accompanied by vomiting and collapse, which still further con-

fuse the diagnosis. Very little dependence can be placed on the localization of the pain. Usually it is most severe in the upper abdomen, and is deeply seated. From this position it may radiate into either hypochondrium, to the back or the lower abdomen.

Pain may be present before there is any definite tumor or its appearance may be delayed until the cyst has reached a large size.

The relation between eating and the occurrence of pain is usually indefinite, but at times it occurs only after eating and may be associated with vomiting.

Vomiting depends on two factors, the severity of the pain and the interference with gastric function. It is almost always a concomitant symptom when pain is severe, and it has a close relationship to the occurrence of exacerbation. Vomiting is also a constant feature in those cases where the growth of the cyst directly compresses the stomach. It may be the delayed vomiting of obstruction of the pylorus, or reflex vomiting from gastric irritability.

Pressure Symptoms.—The grouping of these symptoms depends on the direction of growth of the cyst. Pressure on the stomach causes indigestion, flatulence, discomfort after eating, anorexia and finally vomiting.

Jaundice may be due to direct pressure on the common bile-duct or to associated pancreatitis or gall-stones. Constipation results from pressure on the colon and in a few recorded cases this has gone on to actual obstruction.

More remote results of pressure are ascites from obstruction of the portal vein, œdema of the lower limbs from obstruction to the inferior vena cava, and hydronephrosis from pressure on the ureter.

Functional Disturbances.—The presence or absence of functional disturbances depends on the original cause of the cyst formation and on the degree of destruction of pancreatic parenchyma. If the cyst is caused by or associated with chronic pancreatitis the presence or absence of the signs of pancreatic indigestion depends on the extent of involvement. These signs, steatorrhœa, azotorrhœa, bulky pale stools, the presence of the Cammidge reaction in the urine, etc., are evidence of pancreatitis and not

of pancreatic cyst, but their presence in conjunction with an upper abdominal tumor having the physical characteristics of a pancreatic cyst would be strongly corroborative evidence. Occasionally glycosuria is present.

The absence of signs indicating pancreatic insufficiency does not rule out a diagnosis of cyst of the pancreas, but simply means that there has not been sufficient destruction of glandular parenchyma to interfere with its function.

Loss of weight and strength is nearly always an accompaniment of large cysts and may be well marked even in those of moderate size. It results from various causes, notably from interference with pancreatic digestion, vomiting and gastro-intestinal disturbances and possibly from obscure metabolic changes.

Physical Signs.—The physical signs depend on the presence, direction and rapidity of growth of a cystic tumor originating in the upper abdomen between the ensiform and umbilicus. In the majority of instances the tumor occupies the middle line and projects to the left side more often than the right. Unless too small to reach the surface, the recognition of a rounded, smooth cystic tumor is easily demonstrated by abdominal palpation. The physical signs vary with the direction of growth from the point of origin.

Körte made *three classes of pancreatic cysts according to the direction of growth.* In addition there are cases that do not fit into any of these groups. The direction of growth is influenced by the origin of the cyst in relation to the reflections of peritoneum from the pancreas. Dilatation of the stomach and colon with air shows the relation of a cyst to these viscera. The degree of dilatation influences the amount of contact between the tumor and the abdominal wall.

1. *The first of Körte's groups* comprises those tumors that grow forward between the transverse colon and the stomach, displacing the latter upward and the former downward (Fig. 28). The amount of displacement depends on the size of the tumor and the amount of air in the viscera. Cysts following this line of growth arise from the anterior surface of the head or body of the pancreas or are pseudocysts formed in the lesser peritoneal cavity.

2. *The second group* includes growths from the upper part of the anterior surface of the head or body of the pancreas which

grow forward below the liver and above the lesser curvature of the stomach, displacing the latter downward (Fig. 29). This direction of growth is prone tc occur in those patients subject to gastroptosis.

3. *The third group* comprises cysts from the tail of the pancreas. This lies on the left of the duodeno-jejunal junction and

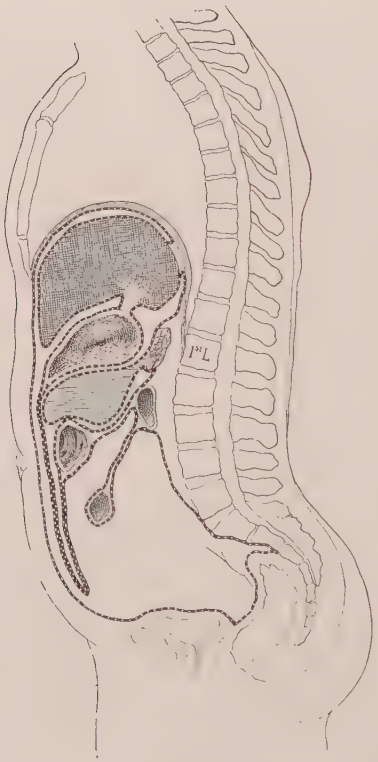

Fig. 28.—Cyst of Pancreas Presenting beneath the Gastro-colic Omentum.

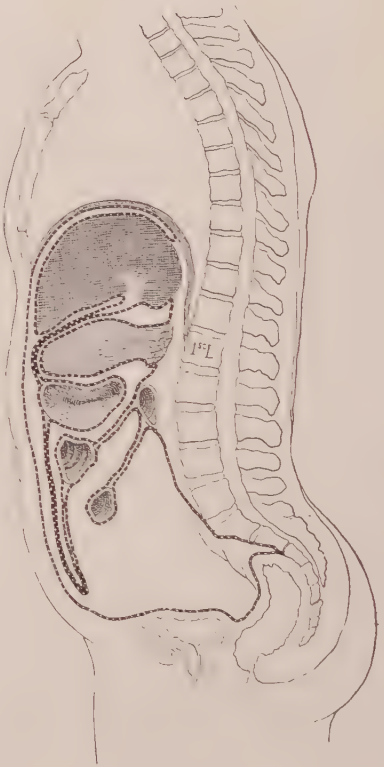

Fig. 29.—Cyst of Pancreas Presenting beneath the Gastro-hepatic Omentum.

cysts developing here grow into the transverse mesocolon and displace the colon in one of three ways, upward, downward or directly forward. If the colon is displaced downward the tumor grows toward the left hypochondrium simulating a splenic growth. A cyst that comes out below the colon or pushes it directly forward, grows toward the midline forming a tumor of the middle abdomen.

Its relation to the colon is readily determined by distending the latter (Fig. 30).

The line of growth toward the *left hypochondrium* may also be taken by cysts arising from the body or tail of the pancreas above the reflection of the transverse mesocolon.

Retroperitoneal growth into *either flank* may occur in cysts arising from the posterior surface of the pancreas. These cysts resemble tumors of the kidney or suprarenal

Growth into the layers of the mesentery resembling a mesenteric cyst; growth forward from the head of the pancreas below the reflexion of the transverse mesocolon below the hepatic flexure of the colon, resembling a tumor of the cæcum, ascending colon or right kidney; and growth through the foramen of Winslow into the general peritoneal cavity have all been reported, but the majority of cases fall into one or another of Körte's three main groups.

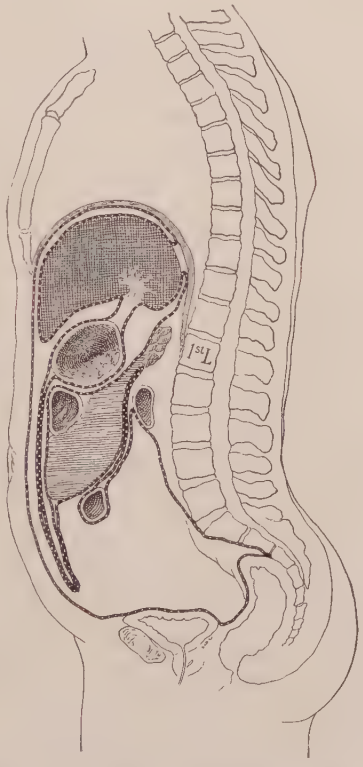

Fig. 30.—Cyst of Pancreas Growing into the Transverse Mesocolon.

Mobility of an upper abdominal tumor does not exclude a pancreatic origin, and some cysts, particularly those that arise from the tail of the pancreas, often are freely movable.

Pancreatic tumors as a rule *transmit the pulsation of the aorta*. This transmission ceases when the patient is in the knee-chest posture.

Disappearance of a cyst may be caused by rupture the result of an injury or exploratory puncture for diagnosis. Several cases have been reported in which the cyst disappeared as the result of discharging its contents into the bowel by way of the pancreatic ducts, or by means of a fistulous opening. This disappearance

is followed by profuse diarrhœa of material resembling cyst contents or saliva. These cysts may refill.

Diagnosis.—From the above description it is evident that there are no pathognomonic symptoms or physical signs of pancreatic cyst. A history of epigastric injury is often an aid in diagnosis. Even at operation it is impossible to differentiate true from false cysts as their appearance and growth are so similar.

Diagnosis depends largely on physical signs and these may resemble those of growths of the liver, kidney, suprarenal, spleen, mesentery or ovary.

As a rule, the *history*, the *relation existing between the stomach and colon*, possibly the accompanying symptoms and signs of *pancreatic insufficiency*, make the diagnosis fairly certain in the majority of cases; but in those exhibiting eccentric forms of growth, particularly if unaccompanied by any symptoms referable to the pancreas, the diagnosis always is more or less uncertain.

Differential Diagnosis. *Mesenteric and Omental Cysts.*—As cysts of the pancreas may occupy either of these positions and are also at times freely movable, it is obviously impossible to differentiate certain cases.

Kidney and Suprarenal Cysts.—The resemblance of these growths to retroperitoneal pancreatic cysts is sometimes very close; and if urinary findings, ureteral catheterization, renal symptoms, X-rays and examination of the fæces give no clue the diagnosis must be doubtful. Tympany behind the axillary line should indicate a pancreatic rather than a renal growth

Cysts of the Liver.—Almost without exception these are echinococcus cysts, but they may closely resemble in physical signs pancreatic cysts growing forward between the liver and stomach. Inflation of the stomach might aid in diagnosis as it is much more likely to obscure a pancreatic than a liver tumor. Obtaining fluid by puncture should ensure a correct diagnosis, but this is attended with considerable danger. Exploratory section is much safer and more certain.

Ovarian Cysts.—It should be the rarest possible occurrence to confuse ovarian and pancreatic cysts after careful bimanual examination, under anæsthesia if necessary. The presence of a normal uterus, tubes and ovaries in one, and in the other the recognition of a pedicle, with possible displacement of the uterus

by traction or pressure and the difference in history make confusion of the two conditions very unlikely.

Enlarged Gall-bladder.—Close contact with the abdominal wall, respiratory movement if non-adherent, dullness continuous with the liver dullness, direction of growth, and the history serve to differentiate a distended gall-bladder from a cyst of the head of the pancreas even if accompanied by jaundice.

Cysts of the Spleen.—The history, direction of growth, physical signs, particularly the relation to the stomach, colon, left rib margin and area of normal splenic dullness serve to make confusion of these two very unlikely.

Prognosis.—Pancreatic cysts may persist for years without causing any symptoms except enlargement of the abdomen. These cases, however, are exceptional.

As a rule the course is progressive, symptoms becoming more pronounced with increase in the size of the tumor. Pressure symptoms become marked, interference with pancreatic function causes loss of weight and strength, and later probably diabetes.

Spontaneous or traumatic rupture may occur at any time and cause death by shock or peritonitis. Suppuration is not very infrequent. W. W. Ashhurst successfully evacuated two quarts of pus by pancreatotomy.

Sudden enlargement accompanied by symptoms of shock indicates hemorrhage into the cyst. Of 160 operations for pancreatic cyst mentioned by Robson and Cammidge, recovery ensued in 140 cases, though eight of these patients died within a few weeks or months. Of thirteen patients under their own care eleven recovered after operation. Among eleven operations for pancreatic cyst by the senior author, there was one death, a mortality of 9 per cent. Four other patients have been under his care, in whom the diagnosis of pancreatic cyst was not confirmed by operation or autopsy: two of these patients went home somewhat improved in health, and two died before operation was permitted.

Treatment.—Medical treatment has no effect on staying the progress of the disease. Aspiration is contraindicated because of the danger of peritonitis or perforation of one of the large vessels in the wall of the cyst.

Complete extirpation is only occasionally possible, usually in those cases where the cyst arises from the tail of the pancreas

or where it grows forward between the layers of the mesocolon. Excision is more difficult and has a higher mortality than incision and drainage and is nearly always impossible because of adhesions (Richardson). It was successful in the case of the patient whose history is detailed below.

Incision and drainage (marsupialization) is the most suitable operation in the majority of cases. The incision is made over

Fig. 31.—Cyst of Pancreas Removed Entire with Pedicle and Some Adjoining Healthy Pancreatic Tissue. (*From a patient in the German Hospital.*)

the most superficial portion of the growth and a rubber tube inserted into the cyst cavity after evacuation of the contents. The tube is held in place by catgut sutures and leakage guarded against by a purse-string catgut suture tied after inverting the drainage wound and tube.

Occasionally drainage through the loin is easier than through the abdomen.

The cyst wall should be fastened to the abdominal wall but not to the skin.

The presence of pancreatic juice in the drainage fluid indicates a direct connection between the pancreas and the cyst cavity. Care must be taken to avoid excoriation of the skin by the pancreatic juice in the discharge. Ointments with a mineral rather than an animal base are recommended since the latter is digested by the discharge. As a rule granulation tissue gradually obliterates the cavity and it closes completely, but occasionally a small fistula may persist for years. Antidiabetic diet, as recommended by Wohlgemuth, hastens the closure of the fistula (page 311).

Cyst of Pancreas; Excision. Recovery.

J. M., aged 17 years. Admitted to German Hospital Dec. 6, 1912.

Chief Complaint.—Pain, sharp and constant, in epigastrium; anorexia, constipation.

Previous Medical History.—Negative.

Family History.—Mother and father living and well. Four brothers and five sisters living and well. Two brothers died in infancy.

Social History.—Eats and sleeps poorly. Bowels irregular. Denies venereal infection. Does not smoke or drink.

Present Illness.—For past seven years has had a gnawing, constant pain in stomach just to left of midline, 4.5 centimetres below the ensiform cartilage, and radiating to the back. It was made worse by eating—the greatest pain coming on one-half to one hour after eating his meals. One year ago the pain became more severe and he is now unable to work. He cannot sleep or eat anything but soft and liquid diet. November 16, 1910, he was operated on in another hospital for acute perforative appendicitis, he made due recovery, but his stomach pain persisted in spite of it. He has never vomited food or blood. Never has passed blood by his bowels, but has been constipated. His pain is present always, feels as if he had a boil on the coating of his stomach. He has no distention, no rigidity and peristalsis is fair.

Physical Examination.—Anæmic Italian boy in great pain. Eyes and ears negative. Lips pallid. Tongue, clear, fissured. Teeth, good. Chest, fair development and expansion. Heart and lungs, surgically negative. Abdomen, muscular, seems wasted. No distention or rigidity. Peristalsis good. Sharp, gnawing, constant pain increasing one-half to one hour after eating, present 4.5 centimeters below the ensiform cartilage to the left of midline. It is aggravated on light palpation. It seems evident that there is an ulcer present. No pain in right iliac fossa. No palpable masses. No herniæ. No adenitis. Genitalia and extremities, negative. Blood pressure —systolic 110, diastolic 80.

Operation.—Dec. 9, 1912. Dr. Deaver. Upper right rectus incision. Peritoneum opened. Adhesions found between the great omentum and parietal peritoneum. Liver adherent to the parietal peritoneum. Peritoneum clamped

to towels. Gall-bladder found normal and connected by adhesions to the hepatic flexure of the colon and the gastro-colic omentum. Adhesions also found between the omentum and the duodenum. Pancreatic lymph-nodes were enlarged. Pylorus patulous. Adhesions between the jejunum and the under surface of the transverse colon. Lesser peritoneal cavity opened through the gastro-colic omentum and mass found in the tail of the pancreas. Mass found to be a cyst the size of a small lemon in the tail. The cyst excised (Fig. 31). Rubber dam drainage. Opening in the transverse meso-colon and the gastro-colic omentum closed with single iodine gut. One piece of rubber dam to the stump of the pancreas. Wound closed to drainage.

Recovery was uneventful, and patient was discharged January 18, 1913.

REFERENCES.

Ashhurst, W. W.: Medical News, 1894, lxiv, 377.
Connolly: Lancet, 1911, i, 803.
Klebs: Hanb. d. path. Anat., Berlin 1869, S. 548.
Körte: Die Chir. Krankh. d. Pankreas, Deutsche Chir., Lief. 45d., Stuttgart, 1898.
Lazarus: Beitr. zur Path. u. Therap. der Pankreaserkrankungen, Berlin, 1904.
Jordan Lloyd: Brit. Med. Jour., 1892, ii, 1051.
Michel and Gross: Rev. de Gyn. et de Chir. Abd., 1904, viii, 45.
Railton: Brit. Med. Jour., 1896, ii, 1318.
Richardson: Bost. Med. and Surg. Jour., 1895, cxxxii, 280.
Robson and Cammidge: The Pancreas, its Surgery and Pathology, Philadelphia, 1907.
Senn: Amer. Jour. Med. Sc., 1885, ii, 17.
Shattuck: see Richardson: Trans. Amer. Surg., Assoc., 1882, i, 211.
Stieda: Centr. f. path. Anat., 1893, iv, 449.
Thiroloix: Archives de Physiol., 1892, iv, 716.
Virchow: Berlin klin. Woch., 1887, xxiv, 248.

CHAPTER VIII.

SURGERY OF THE SPLEEN.

Anatomy.

Position.—The spleen occupies a position in the posterior portion of the left upper abdomen behind and slightly to the left of the cardiac end of the stomach. Its long axis is nearly parallel to the course of the ribs. Its posterior boundary is at a point one and one-half to two inches external to the vertebræ, and it extends to the mid-axillary line anteriorly. Its upper end is opposite the spine of the ninth dorsal vertebra, and its lower end is at the level of the first or second lumbar vertebra. The phrenic surface lies beneath the ninth, tenth and eleventh ribs.

Surface Anatomy.—The normal area of splenic dullness extends in the mid-axillary line longitudinally from the ninth to the eleventh rib, and transversely from the mid-axillary to the posterior axillary line.

Appearance, Size and Shape.—The spleen is of a dark red or purplish color and has an average weight of about 195 grams. The size varies somewhat, being greater at the height of digestion as a result of congestion. The shape depends largely on the surrounding viscera and is best ascertained by hardening *in situ*, by which means one inconstant and three constant surfaces are presented for study. These surfaces are named from the organs with which they are in contact.

The *phrenic surface* is the outer and posterior convex surface lying beneath the diaphragm. It ends in front at the anterior border, which is particularly well marked below the level of the hilum and contains one, two or three notches, a diagnostic sign in enlarged spleen. The anterior border separates the phrenic and gastric surfaces.

The *renal surface* rests against the anterior portion of the upper end of the left kidney and suprarenal capsule. This surface does not extend as high as the phrenic surface. The tail of the pancreas is sometimes in contact with this portion of the spleen, and some-

times with the gastric surface. The renal and phrenic surfaces are separated by the rounded posterior border of the spleen.

The *gastric* or *anterior surface* is concave and is in contact with the fundus of the stomach. This surface contains the hilum, a fissure for the vessels, and at its lower portion is in contact with the splenic flexure of the colon, unless there is a basal surface.

The *basal surface* when present is a small flattened area on the inferior pole of the spleen resting on and being supported by the splenic flexure of the colon.

Blood-vessels.—The *splenic artery* is a branch of the cœliac axis. It is relatively very large for the organ it supplies, and its course is quite tortuous. At the level of the tail of the pancreas it runs foward in the lieno-renal ligament, to break up into several branches which enter the hilum one above another and anterior to the veins. These vessels ramify through the connective-tissue trabeculæ of the spleen and do not anastomose. The branch that enters near the upper pole of the spleen is given off first from the main trunk and supplies several small branches to the stomach (vasa brevia). The gastro-epiploica sinistra is given off from the splenic artery just before it divides into its terminal branches.

The *splenic veins* are formed in the fibrous trabeculæ. They join to form several large branches which emerge from the hilum and unite to form the splenic vein behind and below the artery. The splenic and superior mesenteric veins unite to form the portal vein. This fact accounts for the splenic congestion and enlargement associated with cirrhosis of the liver.

The *lymphatics* emerge from the hilum and empty into nodes at the tail of the pancreas.

The *nerves* come from the solar plexus and enter the spleen with the arteries.

Ligaments.—The spleen is enveloped in peritoneum except where the ligaments meet at the hilum to form the pedicle. These ligaments are folds of peritoneum which transmit the blood-vessels.

When the surgeon's hand is introduced through an abdominal incision and passes over the anterior surface of the stomach to the fundus, it encounters the spleen. The fold of peritoneum thus felt, which joins the spleen to the stomach, is the *gastro-splenic ligament.* In it run the *vasa brevia*, branches of the splenic artery to the fundus of the stomach. If the surgeon carries his hand still

further to the patient's left, it will pass between the spleen and the inferior concave surface of the diaphragm. The fold of peritoneum that arrests the fingers as they pass behind the spleen toward the spinal column, is the *lieno-renal ligament.* If one hand is placed in this situation, and the tips of the other fingers are placed anteriorly on the gastro-splenic ligament, the pedicle of the spleen, with all its contained vessels will lie between the two hands. If the fingers are passed up over the external convex surface of the spleen to its upper pole they will here encounter the suspensory ligament of the spleen the *lieno-phrenic ligament*, which binds it to the diaphram. If this is severed, the spleen may be drawn down from beneath the diaphragm and sometimes may be brought into the abdominal incision.

Relations.—From the description given above it is seen that the spleen is in relation with the following structures: *Externally* and above with the diaphragm, and above this with the pleura, the lung, and the ninth, tenth and eleventh ribs. *Anteriorly* with the stomach. *Internally* with the left kidney and suprarenal capsule. *Inferiorly* with the splenic flexure of the colon.

It is important to remember these relations to surrounding organs when dealing with an injury of the spleen since, especially in cases of penetrating wounds, one or more of the surrounding structures may be injured.

Anatomical Anomalies.—*Accessory spleens* are of two kinds, those consisting of true splenic tissue, and those having the structure of hæmolymph glands. The former are true accessory or supernumerary spleens; usually they are joined by connective tissue to the anterior border of the spleen from which they have been cut off during fœtal life. Occasionally these masses of splenic tissue are found free in the great omentum, in the transverse mesocolon or in the gastro-splenic omentum near the hilum of the spleen. Their significance is unknown. Accessory spleens resembling hæmolymph glands have the same distribution as the true accessory spleens, and in addition are said sometimes to be found within the tail of the pancreas. Usually they are about the size of a pea, and vary in number from fifteen to twenty, although much larger numbers have been reported.

Congenital absence of the spleen, is excessively rare, but has occurred.

Variations in Size.—The spleen may be very small, no larger than a walnut, and all variations from this to the normal size occur.

Lobulated and *abnormal shape* has been noted occasionally, but appears to have no surgical interest.

Movable Spleen is a pathological condition and is described at page 396.

REFERENCES.

Davis, G. G.: Applied Anatomy, Phila., 1910.
Piersol: Human Anatomy, Phila., 1907.

Physiology.

Various functions have been attributed to the spleen, but our knowledge of none of them is very definite. Some theories are supported by a certain amount of experimental evidence while others owe their acceptance to the fact that they have not yet been disproved. The functions of the spleen are best studied by considering first the results of its removal.

The results of splenectomy clearly demonstrate that the functions of the spleen, whatever they may be, readily are assumed by other organs. The changes following excision of a normal spleen can be studied only by animal experimentation, although a certain amount of useful information is obtained after splenectomy in human beings for injury to the normal organ. In the latter cases, however, the organism has to overcome the effects of severe injury and hemorrhage, as well as the loss of the spleen, and the various factors are hard to differentiate. Noguchi has recently recorded the removal of a normal spleen because of its intimate adhesions to a lipoma which was being excised. The patient was a man forty-two years of age, and his blood did not return to normal for five or six years after the splenectomy; at first there was a decrease of the polynuclear leukocytes, and their place was taken by lymphocytes and eosinophile cells. The results of splenectomy for disease of the spleen give no reliable information, as the splenic function practically has disappeared and compensation has occurred before operation.

The *blood changes* following excision of the spleen may be summarized as follows:

Diminution of the red blood cells.
Disproportionate decrease in hæmoglobin.
Leukocytosis.
Eosinophilia and lymphocytosis.

These changes are transitory reaching their maximum in a few weeks or months; a gradual return to normal then occurs. But lymphocytosis and eosinophilia may develop late and persist for some time. No conclusions of any practical value can be drawn from these changes. Infection is responsible for many of the abnormal results of splenectomy in human beings.

The removal of a healthy spleen or one the site of an acute injury, results in a marked *diminution in the number of red blood cells and in the amount of hæmoglobin.* Laudenbach attributes this to the loss of the supposed function of hæmatopoiesis, which is disputed by most authorities. Warthin, on the other hand, concludes that it is due to the increased activity of the hæmolymph-nodes and hyperplastic lymph-nodes, which activity may exceed in its extent the normal hæmolytic function of the spleen, and thus may cause excessive destruction of erythrocytes.

The Blood-forming Function of the Spleen.—The fœtal spleen manufactures *red blood cells* but in spite of Laudenbach's theory, mentioned above, there is no direct evidence to show that this function is normally maintained in post-uterine life.

In cases of *severe anæmia* there are in the spleen collections of cells resembling myeloid tissue, a fact which strongly suggests that, in these conditions, it is actively engaged in the formation of red blood cells (Meyer and Heineke). The administration of pyridin to rabbits results in the formation of areas in the spleen which closely resemble the collections of hæmatogenetic cells in the normal rabbit embryo. Pyridin causes anæmia by great destruction of blood and its mode of action probably resembles that of the cause of primary pernicious anæmia (Morris).

In a case of *splenomegaly with sclerosis of the bone marrow,* reported by Donhauser, the spleen contained islands of what he considered active hæmatoplastic tissue, the bone marrow having lost its bone-forming function as a result of sclerosis.

These observations are sufficient evidence to warrant the assumption that in certain pathological conditions the spleen reverts to its fœtal blood-forming function. That the *regeneration*

of blood after hemorrhage does not depend on the spleen to any extent is shown by the fact that regeneration takes place as quickly in splenectomized as in normal animals (Freytag).

While the red cell-forming function of the spleen is disputed, there is little reason to doubt that this organ is concerned in the formation of *lymphocytes*, in common with the other lymphatic structures of the body.

Hæmolysis and Blood Cleansing.—There is no direct evidence to prove that the spleen is a hæmolytic organ, though this is generally believed. It contains large phagocytes in which are disintegrating red cells or particles of pigment, as well as pigment free in the pulp. This pigment deposit and red blood cell destruction are greatly increased in certain severe anæmias. There is also free in the spleen a large percentage of organic iron. These facts suggest that the spleen either destroys the erythrocytes, or, more probably, acts as a mechanical filter and takes up the remains of the red blood cells, causes their disintegration and again puts the iron into solution.

Metabolic Functions of the Spleen. *Organ of Iron Metabolism.*—Experiments show that the total elimination of iron in splenectomized but otherwise normal dogs is considerably greater than in dogs with spleens. This relation is maintained whether the dogs are starved or well fed on meat. From these facts it may be concluded that the spleen is an organ of iron metabolism, serving to preserve for the use of the organism the iron which is liberated by the metabolism of the body.

Influence on Growth.—Grossenbacher concludes from the results of experiments on puppies, that the spleen exercises no appreciable influence on growth, differing therefore from the thyroid and thymus in that its presence or absence does not definitely influence the course of life.

Protection of the Organism against Infection.—Hubbard, after reporting his own experiments on the resistance of guinea-pigs to staphylococcus pyogenes aureus infection after splenectomy, and reviewing five other communications, concludes "that the removal of the spleen does not alter, practically, the individual's susceptibility to infection and that its functions in this respect, if they do actually exist, on its removal are readily taken up by other organs."

Almost nothing is known of the functions of the spleen and the cause of its enlargement in various infectious diseases and intoxications. This enlargement is supposed to indicate some protective action but definite knowledge is lacking.

Manufacture of an Internal Secretion.—There is a theory, supported by the experimental work of Schiff, Herzen and others, that the spleen elaborates an enzyme which acts on the trypsinogen contained in the pancreas and converts it into trypsin. That this function, if it does exist, is of little moment is demonstrated by the results of experiments with the fresh pancreatic juice of splenectomized animals. It was shown that this contained trypsin in an active form (Pawlow).

In common with various other organs, the liver, pancreas, lungs, etc., the spleen contains a ferment; this is known as adenase, and is capable of converting adenin into hypoxanthin. Its significance is unknown.

Uric acid is present in the spleen and it has been suggested that it is formed as a result of proteid metabolism, but Chittenden and Mendel as the result of their experiments say, "there is no evidence that the spleen exerts any special influence on either carbohydrate or proteid metabolism in general."

Movements of the Spleen.—The nerves supplying the spleen are derived from the sympathetic; their stimulation causes contraction; and their section causes expansion of the spleen.

Roy has shown that the spleen of the dog contracts and relaxes rhythmically about once a minute. It is supposed that these contractions keep up the circulation and make it independent of the general blood pressure. After a meal the spleen slowly expands, reaching its maximum size in about five hours, and then slowly returns to normal. Relaxation of the muscles in the trabeculæ and vasodilatation are supposed to cause this enlargement.

The relation between these movements and the function of the spleen has not been discovered.

REFERENCES.

Donhauser: Bull. Ayer Clin. Lab. Penn'a. Hosp., 1908, v. xlvi.
Freytag: Arch. f. d. ges. Physiol., 1908, cxxii, 501.

Freytag: Folia Hæmatolog., 1908, v. 90, 1910, xi, 33.
Hubbard: Bost. M. and S. Jour., 1909, clx., 743.
Laudenbach: Centr. f. Physiol., 1895, ix, 1; Arch. de Phys. Normale et Path., 1896, 5eS., viii, 693; 1897, ix, 200.
Morris, R. S.: Johns Hopkins Hosp. Bull., 1907, xviii, 200.
Noguchi: Berl. klin. Woch., 1912, xlix, 1839.
Opitz: Proc. Soc. Exper. Biol. and Med., 1908–09, vi., 95.
Roy: Jour. of Physiol., 1880–82, iii, 203.

General Consideration of Enlargements of Spleen.

Most of the conditions which are of surgical interest cause an enlargement of the spleen; and it is convenient to consider the physical signs of such enlargements in this place, before discussing the different diseases of this organ.

As the spleen enlarges it emerges from beneath the ribs about the level of the ninth costo-chondral junction. Further enlargement follows the same line, and an hypertrophied spleen always is in the left abdomen until it reaches the level of the umbilicus. Further enlargement carries the splenic tumor across the middle line as well as downward on the left side toward the pelvic brim. The physical signs vary with the degree of enlargement.

Inspection.—If the abdominal wall is thin the enlarged spleen, particularly its lower border, may be seen moving with respiration below the costal margin. Otherwise inspection reveals only undue prominence of the left hypochondrium over the region occupied by the tumor. The enlargement may be so enormous that the whole abdomen is distended, but careful inspection usually shows this to be more marked on the left side. If the lower pole reaches the pelvis the tumor does not move with respiration.

Palpation.—The characteristic features of an enlarged spleen are its close apposition to the abdominal wall, a sharp inner border, and most important of all, interruption of its inner border by one, two or three notches. These are all readily felt if the organ comes out from under the rib margin to any extent. Splenic tumors always grow *forward;* they never produce fullness in the loin. Unless the spleen is anchored by peritoneal adhesions, it moves freely with respiration. Another very characteristic feature is continuance of the tumor up under the rib margin, closely applied to the abdominal wall. It is possible to insinuate

one's hand between the ribs and the upper border of all but the very largest abdominal tumors, but splenic tumors unless prolapsed are too closely applied to the abdominal wall to permit this. Occasionally when there is marked perisplenitis a friction rub may be felt as the spleen moves with respiration, and adhesions may obscure the notches and twist the spleen so as to obliterate the sharp anterior margin. With the exception of these notches, an enlarged spleen usually is smooth and firm, although in certain infectious diseases it is sometimes too soft to be felt. The enlargement may be so slight that the edge is just to be felt with deep inspiration.

Percussion.—An enlarged spleen is dull to percussion up to the seventh or sixth rib or even higher in the mid-axillary line. The colon is displaced first downward, and later lies behind the enlarged spleen, so that any resonance due to it will be in the flank or loin.

Auscultation.—In the majority of splenic tumors auscultation is negative. If there is any perisplenitis there may be audible a to-and-fro friction rub. Very occasionally there is an audible murmur, hæmic in origin, due to venous dilatation. In obscure cases it is well to outline the enlarged spleen by auscultatory percussion.

REFERENCES.

Cumston: Trans. Amer. Assoc. Obst. and Gynec., 1907, xix, 270.
Enlargement of the Spleen, The Hospital, 1908, xliv, 495, *et passim;* xlv, 12, 64.
Rogs: China Med. J., 1908, xxii, 24.
Warrington: Liverpool Med.-Chir. J., 1908, xxix, 74.

Differential Diagnosis.—Splenic enlargements have to be differentiated from various kinds of abdominal tumors, those in and about the kidney causing the greatest difficulty.

Pancreatic Cysts.—For physical signs and diagnosis see page 375.

Kidney Tumors.—New growths of the kidney scarcely ever come into close contact with the anterior abdominal wall, and even when they do they also cause marked bulging of the loin.

Palpation.—Kidney tumors have a rounded contour with no sharp, notched anterior border. They cause bulging and increased

resistance in the loin when they are large enough to simulate splenic enlargements. The range of motion is much less. The anterior surface slopes away from the abdominal wall as it approaches the rib margin and, except in the very largest tumors the hand can be insinuated between the costal margin and the upper portion of the tumor. An enlarged spleen is closely applied to the abdominal wall, at its upper pole, and the hand cannot be passed above it. A renal tumor seldom is so large that it crosses the median line, but this not unfrequently occurs in cases of splenic enlargement.

Percussion.—The descending colon overlies the anterior surface of the kidney and is pushed forward when the kidney enlarges. This causes an area of resonance over the abdominal surface of the tumor, while there is dullness in the loin. The reverse is true in splenic tumors. But very occasionally the growth of a renal tumor pushes the colon outward instead of forward and the area of resonance is in the flank as in splenic tumors; in such cases the surgeon must rely on the results of palpation and on tests of the renal functions, including catheterization of the ureters, etc.

Auscultation is negative.

Other symptoms and physical signs are of importance. The surgeon should not forget that tumors of the kidney are not always accompanied by characteristic renal pain, and that acute attacks of perisplenitis may cause paroxysmal pain much resembling renal colic. The most important differential signs are obtained by cystoscopy, catheterization of the ureters and careful examination of the urine. In certain cases of splenic enlargement a differential bloo dcount clears up the diagnosis.

Suprarenal growths as a rule have the same physical signs as kidney tumors, except that the colon is very often pushed downward instead of forward. Hæmaturia frequently is present as a result of infiltration of the kidney by the growth.

Perinephric Abscess.—Apart from the evidences of suppuration, the physical signs resemble those of enlarged kidney more closely than they do those of splenic enlargement. The urinary and cystoscopic findings depend on whether or not the kidney substance is involved.

Ovarian Tumors.—Several cases are on record where a displaced spleen has been mistaken for an ovarian tumor, the mistake being recognized only after the abdomen was opened. When the

spleen is in the pelvis and the notch cannot be felt by vaginal or rectal examination it is readily seen how such confusion might occur. A diagnosis of prolapsed spleen can be made only by feeling the notch in the sharp anterior border. A large ovarian tumor could scarcely be mistaken for a tumor of the spleen enlarging from its normal position. The following characteristics of an ovarian tumor are sufficient to make the diagnosis: The upper border of an ovarian tumor seldom is in actual contact with the left costal margin unless it reaches also to the right costal margin. Ovarian tumors grow upward from the pelvis and the first and most prominent enlargement is in the lower abdomen. They do not move with respiration, and have no sharp border with one or more notches. They extend further across the middle line and cause more symmetrical enlargement of the abdomen. Vaginal examination as a rule shows the tumor in close association with a normal sized uterus, and frequently the pedicle of the cyst can be felt through the rectum. There usually is an area of resonance between the upper border of dullness over an ovarian tumor and the normal area of splenic dullness.

Growths of the Splenic Flexure.—Annular growths of the colon in the neighborhood of the splenic flexure usually are malignant and give rise to symptoms of intestinal obstruction before a palpable tumor develops. Occasionally, however, a diffuse tumor forms in the left upper abdomen before symptoms of obstruction occur, and such a tumor may have to be differentiated from an atypical enlargement of the spleen.

A tumor of the splenic flexure has not the definite shape of an enlarged spleen, with its sharp anterior border showing one or more notches, nor has it the same close apposition to the abdominal wall throughout its extent. It is usually dull to superficial, but resonant to deep percussion. If fixed with adhesions it does not exhibit the same degree of mobility during respiration as does the spleen. If not fixed its position changes to a marked extent with changes in the patient's posture. Sooner or later such a tumor gives rise to symptoms of intestinal obstruction and metastasis, but the diagnosis should be made and operation undertaken before these occur.

Tuberculous Peritonitis.—In the fibrous form of tuberculous peritonitis tumors of various sizes and shapes are found in differ-

ent parts of the abdomen. When such a tumor mass is formed in the left upper abdomen and particularly if it is adherent to the spleen it may closely simulate in general outline a splenic tumor. Attention to the following points serves to distinguish one from the other.

The range of movement in a tuberculous tumor usually is limited by adhesions to the abdominal wall. Although the anterior border may be well defined it seldom exhibits a notch similar to those on the spleen. There usually is an area of resonance between the tumor and the normal area of splenic dullness. Moderate ascites often is present in tuberculous peritonitis, but may also occur in some forms of splenomegaly. Careful search nearly always will reveal other tumors or indefinite areas of consolidation in different parts of the abdominal cavity. The tuberculin reaction is of great value; von Pirquet's skin test is sufficient in children, but in adults the hypodermic injection of old tuberculin gives more accurate results.

Malignant Peritonitis.—In this condition the physical signs and symptoms are very much like those of tuberculous peritonitis, except that it is not likely that the tuberculin reaction will be positive. In most cases other tumor masses are readily discoverable in various portions of the abdominal cavity.

Fæcal Impaction.—Except that it may give rise to a tumor in the left hypochondrium there is very little resemblance between a fæcal impaction and an enlarged spleen. The irregular, indefinite shape, symptoms of temporary obstruction alternating with diarrhœa, pitting on pressure, movability and absence of the usual physical signs of splenic enlargement, serve to make the differentiation comparatively easy.

Sarcoma of the Stomach.—Primary sarcoma of the stomach is rare. When it occurs it is frequently situated at the fundus, infiltrates the whole stomach wall, and may cause a very large tumor in the upper abdomen. As previously noted (Vol. I, page 305) it is accompanied by enlargement of the spleen in about 15 per cent. of cases.

Its distinguishing characteristics are: resonance on percussion over the tumor; change of position during examination and when the patient changes his posture; a position further to the right than is usual with an enlarged spleen, that is, the right border

may be beyond the middle line although the tumor does not extend below the umbilicus; definite gastric symptoms; and, while there is a sharp border, it is not notched as a rule. The course of such a case is rapidly progressive to a fatal termination.

Retroperitoneal tumors are not easily confounded with those of splenic origin. Retroperitoneal growths usually present within the circle formed by the large bowel, offering to percussion a dull area surrounded by intestinal tympany. Inflation of the colon and stomach, examination in the Trendelenburg and knee-chest positions, should be adopted in obscure cases.

Causes of Enlargement of the Spleen.

There are two groups of cases associated with splenomegaly. (1) Those in which the blood changes are distinctive. (2). Those in which the blood changes are not distinctive.

A complete blood examination is essential in every case of splenic enlargement. A positive diagnosis never can be made without it. By this means Group 1 is differentiated from Group 2 with comparatively little difficulty. With the exception of certain cases of malaria to be considered later (see page 428) operative interference is absolutely contraindicated in cases included in Group 1, and therefore they have no place in the consideration of splenomegaly viewed from a surgical standpoint.

Group 1. Cases of Splenic Enlargement with Distinctive Blood Changes.—This comprises most cases of malaria, the leukæmias, pernicious anæmia, splenomegalic polycythæmia, typhoid fever, and kala azar.

Group 2. Cases of Splenic Enlargement without Distinctive Blood Changes.—This comprises two divisions, (*a*) cases in which operative interference is advisable or necessary, and (*b*) those in which it is contraindicated.

The first division, in which *splenectomy may be necessary,* includes:

1. Movable spleen.
2. Cysts.
3. Tumors.
4. Tuberculosis.
5. Abscess.
6. Banti's disease.
7. Traumatic enlargement.

The second division is of surgical interest only from the standpoint of differential diagnosis, since *splenectomy is contraindicated*. It includes enlargements due to:

1. Congestion and inflammation.
2. Infarct and thrombosis.
3. Infectious fevers.
4. Hodgkin's disease.
5. Cirrhosis of liver.
6. Syphilis.
7. Amyloid disease.
8. Congenital syphilis and rickets.
9. Perisplenitis.
10. Pseudoleukæmia.
11. Hereditary and family forms of splenomegaly.
12. Pressure on portal vein by tumors of surrounding organs, liver, gall-bladder, pancreas and stomach.
13. Obstruction of the inferior vena cava above the entrance of the hepatic veins by mediastinal tumors, pulmonary fibrosis, chronic heart disease, etc.

The diagnosis of some of these from surgical conditions is obvious. The others will be considered with the conditions which they simulate.

Movable Spleen.

A movable spleen is also known as a *wandering*, a *floating*, or an *ectopic* spleen. The terms *prolapse* and *splenoptosis* are also applied. A *dislocated* spleen is one which is fixed in an abnormal position.

Ætiology.—Prolapse of the spleen occurs most frequently in women; thirteen of the fourteen cases reported since 1908 were in women. Elongation of the ligaments always is associated with any marked degree of splenic mobility. This lengthening may be congenital or acquired. Of the congenital condition we know nothing. Acquired lengthening sometimes is said to result from trauma, but it is probable that the injury leads to abdominal examination, and that the discovery is then made of a prolapse of the spleen which has existed without symptoms for some time. It is possible, however, that acute displacements may result from rupture of the liagments. Cases of movable spleen which produce no

symptoms usually are discovered incidentally during examination for other conditions. General enteroptosis may include descent of the spleen, without necessarily any increase in the length of the pedicle. Increased weight is a factor in ætiology, although in the majority of cases of splenomegaly perisplenic adhesions and hypertrophy of the ligaments keep the organ in place. Thus the enlarged spleen of leukæmia, splenic anæmia, malaria, etc., usually does not prolapse. Yet of fourteen cases of splenectomy for wandering spleen reported since January, 1908, four were associated with malarial hypertrophy and three with idiopathic enlargement. Elongation of the pedicle to 10 inches is reported and it often is long enough to permit the spleen to drop into either iliac fossa, or into the pelvis.

Pathology.—A movable spleen may vary in size from normal almost to any degree of enlargement. The pathological conditions in such a spleen are of two kinds, those the result of the displacement, and those occurring independently. Among the latter are morbid changes the result of fortuitous diseases such as malaria and leukæmia. These changes are characteristic and well known and need not be considered in connection with the pathology of movable spleen. Those changes produced by displacement are caused by twisting of the pedicle and interference with the blood supply.

Acute torsion occurs suddenly and the twist is tight, causing necrosis of the spleen and thrombosis of the splenic vessels, unless operative interference is immediately undertaken.

Chronic Torsion.—When twisting occurs slowly, the twist is loose and causes only gradual interference with the blood supply resulting in enlargement from congestion of the spleen, hemorrhage into its substance and consequent increase of fibrous tissue. In late cases these changes may be succeeded by sclerosis and atrophy with consequent diminution in size.

Intermittent attacks of twisting have effects similar to those seen in cases of chronic torsion. Such attacks in a wandering spleen usually cause perisplenitis and the formation of adhesions to the structures with which it comes into contact.

The degree of twisting in reported cases varies from one incomplete to four complete turns.

Symptoms.—Simple uncomplicated mobility causes little or

no disturbance and the diagnosis is usually made accidentally during routine abdominal examination. Occasionally indefinite gastro-intestinal symptoms are associated with splenic mobility and may be considered the consequence of such displacement. Intermittent attacks of jaundice and functional disturbances in surrounding organs may result from the traction of an enlarged movable spleen. Digestive disturbances, constipation and even partial intestinal obstruction may result from dragging of the pedicle. But the only definite symptoms directly attributable to prolapse of the spleen are those arising from twists of the pedicle.

Acute Torsion.—The primary and most important symptom of this condition is severe paroxysmal pain and tenderness in the left hypochondrium. The pain may give rise to nausea and vomiting and a certain amount of shock, with increase in the pulse rate. If unrelieved the obstruction to the blood supply causes necrosis of the spleen and the development of localized peritonitis. Death from sepsis or the formation of a localized abscess may result.

Chronic Torsion.—The twist in the pedicle not being so tight as to cause complete obstruction of the vessels, the symptoms are less sudden and severe in onset. Torsion through less than three-fifths of a circumference seldom produces symptoms. Intermittent attacks of pain associated with local tenderness are the only symptoms of moment. These attacks may occur spontaneously or result from some sudden exertion or movement. Attacks of perisplenitis cause the same symptoms and are the cause of the formation of the adhesions that fix the spleen in abnormal positions (*dislocated spleen*). Rectal tenesmus is caused by impaction of a dislocated spleen in the pelvis. Attacks of perisplenitis or twist of the pedicle usually are accompanied by elevation of temperature and increased pulse rate.

Diagnosis.—In uncomplicated cases of movable spleen the organ is not enlarged to any extent and presents well-marked characteristics. It retains its shape, a sharp anterior border, and one or more distinct notches. The normal area of splenic dullness is absent. By putting the patient in the dorsal position, the spleen can be easily pushed up under the left rib margin, where it will remain until the patient changes her position.

Twist of the pedicle causes increase in the size of the tumor, which becomes acutely tender. Rigidity of the abdominal muscles is pronounced but not general, being confined to the area covering the displaced spleen. If there are no adhesions the characteristics of the splenic outline are recognizable, although a diagnosis of movable kidney is frequently made. As a rule the displacement, the swelling and the formation of an inflammatory exudate obscure the physical signs and render diagnosis difficult. Cases are reported in which a dislocated spleen has simulated appendiceal abscess, twisted ovarian cyst, and pus tube. If the spleen is impacted in the pelvis, the correct diagnosis is impossible unless the notch can be felt through the vagina or rectum, and even then the infrequency of the condition will cause a doubt as to the probability of such a tumor being the spleen.

Differential Diagnosis.—In non-adherent cases the only confusion in diagnosis arises from the question of movable kidney, the great frequency of which condition and its similarity in symptoms and physical signs making a mistake pardonable. But careful examination for the splenic notch, attempting to palpate the kidney independent of the abdominal tumor, the absence of the urinary changes which frequently result from a kidney crisis and the different range of mobility in the two organs, are signs to be looked for and given due weight in arriving at a diagnosis. The splenic artery is said to be palpable in cases of movable spleen, but this is not true of the renal artery when the kidney is displaced. Forgetting the possibility of a movable spleen is the most frequent cause of mistake.

The diagnosis of an adherent misplaced spleen usually is impossible, since inflammatory exudate obscures the splenic outline, and the history and physical signs point to inflammation of the organ normally occupying the area affected. Thus a mass in the right iliac fossa, associated with pain, tenderness, rigidity, fever and leukocytosis is more likely to be an appendiceal abscess than anything else. Likewise, if the tumor is in the pelvis it simulates ovarian or tubo-ovarian disease.

Treatment.—For those cases of movable spleen discovered accidentally, no operative treatment need be undertaken. The patient does not suffer from the condition and no harm is likely to arise. For those cases complicated with twists, perisplenitis and

adhesions operative treatment is indicated and consists in splenopexy or splenectomy, preferably the latter (see page 428). There are seventeen operations for wandering spleen reported from January 1, 1908, to December 31, 1911, (fourteen splenectomies, two splenopexies, and one exploratory operation in which nothing was done when the tumor in the pelvis was found to be the spleen). All the patients made uneventful recoveries. Splenectomy sometimes is very difficult on account of the adhesions formed between the spleen and various abdominal organs. G. G. Ross did splenopexy in one patient with ptosis of the spleen, in the German Hospital, but symptoms persisted and splenectomy was eventually required and proved perfectly successful.

REFERENCES.

Blackburn and Craig: Australas. Med. Gaz., 1907, xxvi, 615.
Lahey: Annals of Surgery, 1911, ii, 612.
Sourdille: Gaz. Méd. de Nantes, 1911, xxix, 212.

Cysts of the Spleen.

The two main varieties of cysts found in the spleen are the **parasitic** (chiefly echinococcus) and the **simple,** or non-parasitic. One case of **dermoid cyst** of the spleen was reported by Andral in 1829. The entire subject has been reviewed recently by Fowler.

Echinococcus Cysts.—Among native born Americans echinococcus disease is almost unknown. In 1901 Lyon collected 241 cases of echinococcus disease reported in North America. The nationality of 149 patients was determined and among these there were only two Canadians and one American, the rest being foreigners (136), negroes (10) and undetermined (92). Of the 241 cases the spleen was the seat of disease in nine (3.7 per cent.).

Pathology.—Involvement of the spleen may be primary or by extension from the gastro-splenic omentum. Any portion of the organ may be affected and it may contain more than one cyst, but each individual cyst is unilocular. The size of the cysts varies; very often they are so small as to remain undetected until autopsy, at other times they reach a size which necessitates surgical interference. Small cysts may be destroyed spontaneously and the remains may become calcified. Large cysts may rupture or become

infected. Rupture leads to serve toxæmia and peritonitis, which may be fatal, or to the danger of dissemination of the disease by the escape of living parasites, if the more serious complications do not occur. Infection results in the formation of an abscess, presenting no special characteristics.

Macroscopically and microscopically the cyst presents the same appearance and causes the same tissue reaction in the spleen as in other organs.

Symptoms.—The symptoms are those of simple cyst. The "hydatid thrill" is usually absent, but may be simulated by other conditions.

Differential Diagnosis.—Exploratory puncture seldom is justifiable on account of the danger of toxæmia, infection and the escape of living parasites into the peritoneal cavity. The finding of cyst elements in puncture fluid is pathognomonic but their absence does not exclude the possibility of echinococcus disease. The information obtained by exploratory puncture is not of sufficient value to warrant the risk incurred. A complement fixation test may also be used, as in hydatid disease elsewhere in the body.

Prognosis.—Cysts of large size are always a source of danger. Rupture or infection may occur and are very serious complications.

Treatment.—Splenectomy is the ideal treatment but may be impossible on account of extensive adhesions. Under these conditions excision or resection of the cyst and drainage should be undertaken. Successful results from marsupialization are reported.

Simple or Non-parasitic Cysts. Causes.—Trauma is the most frequent cause of non-parasitic cyst of the spleen and is the only ætiologic factor that is accepted without question. It gives rise to a subcapsular hæmatoma which subsequently may become a serous cyst from deposition of the solid elements.

Blood Cysts.—In addition to trauma, other causes of blood cysts are hæmangeiomata, hemorrhage into serous or lymph-cysts, and hemorrhage into the spleen during the course of infectious fevers, typhoid, malaria, etc.

Serous Cysts.—Serous cysts usually result from hæmatomas, but occasionally their origin is undetermined and several theories have been advanced to account for their development. It is held by some that a cyst may result from degeneration of portions

of the peritoneal endothelium misplaced beneath the splenic capsule during embryonic life. Another theory is that some of the splenic pulp is extruded through a rent in the capsule and that a cyst is developed in the space that is left.

Lymph-cysts.—Lymph-cysts are due to the degeneration of lymphangeiomata or to the occlusion of lymph-vessels and the escape of lymph.

Retention cysts do not occur as there are no tubular glands in the spleen.

Pathology.—*Blood cysts* contain blood and blood remnants. The color varies according to the age of the cyst and the degree of alteration in the blood. *Serous cysts* are characterized by clear fluid contents, of low specific gravity (1003 to 1010), containing no albumen. *Lymph-cysts* contain fluid having the characteristics of lymph; it is clear, straw colored, has a high specific gravity and contains a large amount of albumen. Cholesterin in varying amounts is contained in the fluid of every splenic cyst. When it is excessive the cysts are known as *cholesterin cysts.*

Non-parasitic cysts may be uni- or multilocular, and vary in size from those which are of no pathological significance to those containing 2 gallons of fluid. The cyst walls also vary from those of extreme thinness, to the thickened and calcified walls of old cysts. The cysts are lined with endothelium, but this is not always demonstrable if the walls have undergone degeneration. Any portion of the spleen may be involved at first, but as the cyst develops it very soon becomes subcapsular. Rapid increase usually but not always is due to hemorrhage into the cyst. Huntington reports a case where a lymph-cyst increased from the umbilicus to the pelvic brim in three weeks.

Adhesions may form around the spleen and make operation very difficult. Unlike hydatid cysts, rupture or infection scarcely ever occurs in cases of simple cyst.

Symptoms.—Pain is the most noticeable symptom. It is due to three main causes: the weight of the tumor, stretching of the capsule, or attacks of perisplenitis. Pain from the weight of the tumor varies in degree and may be only a sense of fullness and discomfort. Sudden stretching of the capsule and attacks of perisplenitis cause severe pain. Other symptoms result from dragging on and interference with the function of neighboring organs.

Gastric, intestinal, urinary, respiratory and cardiac disturbances are thus occasioned. In some reported cases the patients have had chills, although the malarial parasite was not demonstrable.

The *physical signs* are enlargement of the left upper abdomen, a palpable, smooth tumor emerging from beneath the left costal margin, and fluctuation. Small cysts cause no symptoms or physical signs and require no treatment.

Diagnosis.—The diagnosis is based on the physical signs, and on the existence of a cystic tumor growing from and intimately associated with the spleen. Cysts of the spleen have been mistaken for pancreatic cyst, movable kidney, cystic kidney, hydronephrosis and ovarian cyst. These conditions have been sufficiently described.

Treatment.—Surgical treatment is indicated whenever the cyst grows rapidly or is of a size sufficient to cause distressing symptoms. Excision of the cyst with repair of the raw area, or splenectomy are the accepted methods of treatment at present. Marsupialization and incision and drainage should be considered obsolete. Successful results have followed all forms of treatment, but splenectomy has been performed oftener than any other operation. There have been twenty-five operations reported, with one death (page 431).

REFERENCES.

Andral: Précis d'Anat. Pathol., Paris, 1829, ii, 432.
Bacelli: Policlinico, 1897, v, 4.
Bazy: Bull. Soc. Chir., Paris, 1909, xxxv, 841.
Bircher: Deut. Zeit. f. Chir., 1908, xcii, 323.
Fowler: Annals of Surgery, 1913, i, 658.
Huntington: Trans. Amer. Surg. Assoc., 1907, xxv, 366.
Johnston: Surg. Gynec. and Obst., 1908, vi, 615.
Lyon: Osler's Modern Medicine, Phila., 1907, vol. iv, p. 735.
Solieri: Deut. Zeit. f. Chir., 1910, cvi, 594.

Tumors of the Spleen.

These are rare. Types of sarcoma are the least unusual, but are more often secondary than primary. Fibroma, chondroma, osteoma, and lymphangioma have also been observed.

The proper treatment is splenectomy.

REFERENCES.

Fowler: Long Island Med. Jour , 1911, v, 265.
Jepson and Albert: Annals of Surg., 1904, xl, 81.

Tuberculosis of the Spleen.

Usually this is a secondary involvement, though a few cases of apparently primary infection have been recorded. The spleen is not much enlarged. The formation of a cold abscess is common. Splenectomy is the only efficient treatment, but seldom can be done if the disease is so far advanced as to make the diagnosis reasonably certain before the abdomen is opened.

REFERENCES.

Garrow: Amer. Pract. of Surg., ed. by Bryant and Buck, N. Y., 1911, vol. viii, p. 68.
Lefas: La Tuberculose Primitive de la Rate., Paris, 1903.

Abscess of the Spleen.

Abscess of the spleen, or **acute suppurative splenitis,** may occur in the course of pyæmia from any cause. In malignant endocarditis, infective emboli lodging in the spleen give rise to abscesses, which are usually multiple and small. Infection of infarcts the result of thrombosis of branches of the splenic artery gives rise to abscesses which may be of sufficient size to occasion definite physical signs. Cases of abscess of the spleen that are amenable to surgical treatment usually have been preceded by some acute infectious fever. Malaria, typhoid, dysentery, influenza, dengue, and relapsing fever have been reported as predisposing causes. Malaria is the most frequent infection preceding the development of splenic abscess. The infecting agent in general infections varies with the original cause but in those cases where the spleen is the only seat of disease the colon bacillus is the organism most often found, although staphylococci are occasionally observed, and it is probable that the typhoid bacillus alone is capable of causing suppuration. The majority of reports do not include bacterial findings. Occasionally the slpeen may be involved in an abscess caused by extension from a neighboring focus of infec-

tion, such as empyema, subphrenic abscess, perforated gastric ulcer, perinephric abscess, etc. Hæmatomas and cysts (simple or parasitic) may become infected with resulting abscess formation. Neugebauer has reported a case following appendicitis.

Pathology.—Pyæmic abscesses are multiple and small and have no distinctive characteristics. Those the result of infection of an infarct usually are single and they always extend to the periphery. The majority of abscesses amenable to surgical treatment (this includes particularly those resulting from infectious diseases) are single and of large size. When they reach the surface of the spleen they excite perisplenitis resulting in the formation of adhesions to surrounding structures. The spleen always is enlarged, due partly to the presence of the abscess and partly to the inflammatory reaction it sets up. In deep-seated abscesses the enlargement is uniform, but when they extend to the periphery the swelling is localized, except in the very largest abscesses with destruction of the whole splenic pulp. The abscess cavity contains pus cells, broken down blood and splenic pulp. The wall is formed by necrotic splenic tissue surrounded by an area of inflammatory reaction. Occasionally the reaction is sufficient to isolate and completely wall off small abscesses so that the contents eventually become inspissated and the area of infection becomes quiescent. Infiltration of the abscess wall with lime salts may occur, and in some cases overgrowth of connective tissue with absorption of the abscess has resulted, leaving a small depressed scar to mark the site of the original lesion.

An infected hæmatoma or cyst has its own pathological anatomy, to which is added that peculiar to abscess formation.

Symptoms.—*Pain* in the left hypochondrium is the first and most constant symptom. It varies in character from a dull ache to severe paroxysms of extreme intensity. Fever, chills and sweats are fairly constant accompaniments, but their significanec may be masked by the original disease, as when an abscess develops in the course of malaria. Occurring in pyæmia, infection of the spleen is indicated by acute pain in the splenic region and aggravation of the symptoms of infection, with the occurrence of chills, fever and sweats. Quite often an abscess develops gradually, with obscure symptoms, and comparatively little pain and fever.

Perforation of a splenic abscess causes symptoms that vary with the site of the rupture. General peritonitis results from perforation into the peritoneal cavity, although this usually is prevented by the presence of adhesions. Vomiting of pus and blood follows perforation into the stomach, empyema a perforation through the diaphragm, and the passage of blood and pus by rectum a perforation into the colon. *Subphrenic abscess* due to disease of the spleen usually is located in the lesser peritoneal cavity. Among forty-four cases of suppuration in the lesser peritoneal cavity, tabulated by Michel and Gross, seven were due to disease of the spleen.

The general systemic disturbances resulting from splenic abscess are those of suppuration anywhere, and their severity varies in individual cases. Hectic fever, leukocytosis, secondary anæmia, gastro-intestinal disturbances, anorexia, loss of weight, etc., occur; but as these may be present in any form of infection they have no localizing value.

Physical Signs.—The physical signs are those of enlargement of the spleen, with tenderness and rigidity more or less localized when the abscess approaches the surface. A circumscribed prominence may be seen and felt in moderate-sized abscesses, but when very large the abscess occupies so much of the substance of the spleen that surface localization does not occur. If the tenderness is not too great, fluctuation may be obtained in certain cases; in others the tumor is soft and doughy in consistence, with irregular indurated edges; and in still others no area of softening can be made out. A friction rub may be heard sometimes when perisplenitis is present.

An abscess which develops toward the deeper surfaces of the spleen will cause no demonstrable localizing physical signs, except enlargement of the spleen.

Diagnosis.—Pain over the spleen is by no means pathognomonic of abscess as it occurs also in perisplenitis, embolism, cysts, enlargements in some acute fevers, etc. Leukocytosis is not a constant factor, both the actual and differential count being normal in many cases. Definite fluctuation is infrequent. There are, therefore, always certain cases with such obscure histories, symptoms and physical signs that diagnosis before operation is difficult or impossible.

Exploratory puncture never should be undertaken because of the danger of peritonitis.

Frank cases of splenic abscess present fewer difficulties. Pain and tenderness over an enlarged spleen during or following some infectious fever, accompanied by chills, fever and sweats and polymorphonuclear leukocytosis, indicate the probable presence of suppurative splenitis. If, in addition, a localized fluctuating swelling develops the diagnosis is certain. In those cases in which neither a local softening nor diffuse fluctuation is demonstrable, rapid and progressive enlargement of the spleen is a valuable sign, and when taken in conjunction with a history indicating infection, is very characteristic.

Careful attention to the previous history, the symptoms and course of the illness, and the physical signs indicate the correct diagnosis in a majority of cases, in fact in all but those whose obscurity renders accurate diagnosis impossible. At the onset, the condition frequently is mistaken for a relapse of the original illness (malaria, typhoid fever, influenza, etc.)

Abscesses that do not cause palpable enlargement are of no surgical significance. Very large splenic abscesses have to be differentiated particularly from perinephric abscess, and from pyo- and hydronephrosis.

Prognosis.—In abscesses of embolic origin the prognosis is that of the original condition, as a rule very unfavorable. Those amenable to surgical treatment are not the result of pyæmia and gives a much more favorable outlook. A number of recoveries following operation are reported. Recovery has occasionally followed spontaneous rupture and discharge, but the abscess usually reforms if the patient survives the rupture. In single abscesses arising from local conditions the prognosis depends on early recognition and prompt operation. Under these circumstances a large percentage of recoveries may be expected.

Treatment.—Incision and drainage is the operation usually undertaken and the one most generally applicable. Under certain circumstances splenectomy may be preferable, as for instance, when the abscess recurs after drainage. As a rule perisplenic adhesions render splenectomy impossible.

Johnston in 1908 collected nine splenectomies for abscess with eight recoveries. Perkins, in 1907, wrote that splenectomy had

been performed successfully ten times for splenic abscess. All reported cases since 1908 have been treated by incision and drainage.

REFERENCES.

Bakshi: Indian Med. Gaz., 1908, xliii, 340.
Belloni and Moschini: Gaz. d. Osped. e del. Clin., 1910, xxxi, 145.
Brady: Virginia Med. Semi-Monthly, 1907–08, xii, 538.
Fauntleroy: Jour. Amer. Med. Assoc., 1911, i, 260.
Gupta: Indian Med. Gaz., 1908, xliii, 260.
Johnston: Annals of Surg., 1908, xlviii, 50.
McGregor: Brit. Med. J., 1912, i, 240.
Michel and Gross: Rev. de Gyn. et de Chir. Abd., 1904, viii, 45.
Neugebauer: Berlin klin. Woch., 1909, xlvi, 102.
Perkins: Trans. Southern Surg. and Gynec. Assoc., 1907, xx, 541.
Peters: Alabama Med. J., 1910, xxiii, 216.
Pitt and Fagge: Practitioner, 1911, lxxxvi, 541.
Riese: Deutsch. med. Woch., 1908, xxxiv, 329.

Banti's Disease or Splenic Anæmia.

This group includes splenic pseudoleukæmia, primary splenomegaly with anæmia, splenic lymphadenoma, idiopathic or cryptogenic splenomegaly, splenomegalic cirrhosis of the liver and primitive endothelioma of Gaucher, although many investigators class the latter disease as a new growth. With this exception it is generally considered that all the other conditions represent different names for, or various stages of the same disease, but a great deal of confusion still exists in regard to the classification of diseases characterized by splenomegaly and anæmia. From a surgical standpoint the conditions here included as stages of or different names for Banti's disease have a strong reason to be grouped together, apart from their symptoms and pathology, as they are all, except splenomegalic cirrhosis, greatly improved if not permanently cured by splenectomy. Cirrhosis is the last stage of Banti's disease and when it appears the time has passsed when splenectomy could have had any effect on the progress of the disease.

Banti's disease was first described by him in 1882, and is characterized by great chronicity and three definite clinical and pathological stages:

1. *Simple enlargement* of the spleen,
2. Enlargement with *secondary anæmia.*
3. *Cirrhosis of the liver* with splenomegaly.

Ætiology.—Our knowlegde of the ætiology of Banti's disease does not rest on a firm basis. Many theories have been advanced but no definite causative agent is known.

Banti's own belief is that the disease is due to an unknown infective agent setting up changes of a non-inflammatory character. This agent is carried by the blood to the spleen and causes degeneration of the splenic pulp and follicles and hyperplasia of the reticulum; the latter is the more conspicuous feature and results in the development of the fibro-adenomatous appearance so commonly seen; the morbific agent, according to Severino, is then carried away from the spleen in the splenic vein, which at autopsy always is found sclerosed. Two theories as to the origin of this toxin are maintained; according to one theory this toxin is supposed to be elaborated in the spleen, which is recognized as the seat of primary disease; the other theory is that the toxin is formed as the result of disordered metabolism elsewhere in the body. It is not improbable that toxins may be formed in both ways. Banti favors the second theory of the origin of the toxin. Osler suggests an autointoxication of gastro-intestinal origin as the starting-point of the disease. Rolleston believes that the spleen is the site of a chronic infective or toxic process which occasions the pathological changes observed there and which subsequently inhibits blood formation and causes secondary anæmia.

Discussing splenic anæmia, Sutherland and Burghard advanced the theory that this condition is a functional disturbance of the spleen and not necessarily an actual disease. They explain the origin of the signs and symptoms as follows: Normally the endothelial cells of the spleen ingest old red blood cells. Loss of vasomotor control of the splenic artery, due to disease of the visceral sympathetic ganglia (Barr), causes overfilling of the spleen, and this results in hyperplasia and increased functional activity. In consequence of this increased activity the endothelial cells destroy both diseased and healthy red blood cells (Harris and Herzog). The quick regeneration of erythrocytes and hæmoglobin after splenectomy indicates hæmolysis and not

diminished blood formation as the cause of the anæmia. The rapid rise in the number of red blood cells suggests that the blood-forming organs, which have been working at high pressure to overcome the excessive detruction caused by the spleen, are able to regenerate the blood very rapidly after the spleen has been extirpated.

The *bacteriology* of Banti's disease is negative. Animal experiments and careful study of operative and postmortem specimens have emphasized the fact that, if it is an infective disease, the causative agent is still unknown.

No connection exists between the *previous medical history* and the occurrence of Banti's disease; syphilis, malaria, other infectious diseases and alcoholism have not been proved to have ætiological significance. Nor is there any evidence to show a *family predisposition* for the disease except in the case of the primary endothelioma of Gaucher. Several observers of the latter disease have reported a family incidence with a tendency to appear in several members of the same generation.

Age.—Cases are reported from six to seventy-two years but the great majority occur between twenty and forty-five years. The age of Banti's fifty patients ranged from twelve to fifty-five years, two occurring before the age of fifteen years, seventeen between fifteen and twenty-five years, fifteen between twenty-five and thirty-five years, eleven between thirty-five and forty-five years, and five between forty-five and fifty-five years. The age of Osler's fifteen patients ranged from twenty to fifty-eight years; that of West's twenty patients from nine to seventy-two years. Sutherland and Burghard report a splenectomy for splenic anæmia in a child six years old. It is probable that many cases begin in childhood and on account of the chronic course of the disease the patient does not come under treatment for some years. The senior author has recently operated on a female child twenty-two months old, with recovery. The differential diagnosis of the splenomegalies of childhood offers unusual difficulties on account of their number, variety and obscure ætiology.

Sex.—The first series of cases of Banti's disease published showed a great preponderance of males, thirteen of Osler's fifteen; nineteen of West's twenty-four, and seven of Lyon's eight cases. More recent reports do not sustain these figures; thirty-two of Banti's fifty patients were females, and of Simonds's collected

forty-eight cases, twenty-five were females and twenty-three males. Torrance collected thirty-six splenectomies for Banti's disease; seventeen of the patients were females, fifteen were males. In four the sex was not mentioned. Practically, therefore, both sexes are about equally affected.

No *race* or *country* is particularly associated with the occurrence of the disease.

Pathology.—The first changes occur in the spleen, and these attain their permanent character before any other pathological alterations appear. Then secondary anæmia develops and finally the changes in the liver and portal system complete the morbid anatomy of the disease.

Spleen.—The spleen enlarges steadily but retains its normal shape and appearance. The average weight is from 1500 to 1750 grammes but enormous hypertrophy is reported at times. The largest spleen in Simonds's collected cases weighed 4500 grammes, and in Bovaird's case of endothelioma the spleen weighed 6250 grammes.

Very commonly there are a large number of perisplenic adhesions, and these may give great trouble at operation, sometimes rendering splenectomy impossible.

The capsule and fibrous tissue of the trabeculæ undergo considerable hypertrophy, but the most marked change is hyperplasia of the reticular fibres without any marked change in the cellular elements. The Malpighian corpuscles are overgrown with connective tissue and atrophied, and in some places have completely disappeared. In addition the whole spleen shows more or less passive hyperæmia. There is also in every case some proliferation of the endothelium of the sinuses. Very often normal areas of splenic tissue with functionally active Malpighian corpuscles remain scattered throughout the substance of the spleen.

The amount of endothelial proliferation varies and may be so great as to cause the characteristic changes known as *primitive endothelioma of the type of Gaucher*. In these cases the spleen as a rule is uniformly enlarged, but in Stengel's case a nodular growth was present. Microscopic examination reveals hyperplasia of the endothelium occurring in large masses. In addition there are areas of necrosis and hemorrhage and overgrowth of the reticular connective tissue. The liver, lymph-nodes and bone marrow also

show these endothelial accumulations. The appearance of the spleen is strongly suggestive of a new growth; but the long duration, the uniform distribution of the areas of endothelial proliferation and the cure of the condition by splenectomy are strong arguments against malignancy. Pathologically, primitive endothelioma is a distinct and definite form of splenomegaly but clinically it has the characteristics of splenic anæmia.

Splenic Vein.—There is always some chronic sclerosing endophlebitis of the splenic veins and in some cases it is excessive, with calcification and stenosis. The portal vein may also be affected.

Liver.—There is no change in the liver until late in the second stage of the disease. From this time on the changes cannot be distinguished from those of Laennec's atrophic cirrhosis.

Symptoms and Physical Signs.—Banti's disease runs an extremely chronic course of from five to twenty-five years.

First Stage.—This is characterized by its insidious onset and long duration. There is gradual progressive and painless enlargement of the spleen, which may be accompanied by a sense of weight and discomfort in the left hypochondrium. The increase in size of the spleen continues until the lower pole is on a level with or below the umbilicus, but when the pelvic brim is reached growth ceases. The splenic tumor in these cases is as big as any except the largest of the leukæmic spleens. There is no connection between the size of the spleen and the occurrence or severity of the symptoms. Splenomegaly is present for three to five years or longer before the onset of the symptoms of anæmia which mark the beginning of the second stage.

Second Stage.—The early symptoms of this stage are those of simple anæmia, pallor, weakness, dyspnœa and palpitation. The grade of the anæmia has no relation to the severity of the disease. With the development of these symptoms the blood usually shows changes of a chlorotic type—diminution of red blood cells and hæmoglobin, with a low color index. The hæmoglobin may be diminished to 50 or even 40 per cent. In addition there is also leukopenia with relative lymphocytosis; the total count may fall to 1000–1500 per cubic millimetre. Leukopenia is very characteristic of splenic anæmia, but in common with diminution in the red blood cells and hæmoglobin is not

necessarily present in all cases. These changes may be very slight or even completely absent even when the symptoms of anæmia are well developed. Later in the disease the blood picture may be profoundly modified by hemorrhages.

Following the appearance of anæmia, changes in the amount and quality of the urine occur. Banti in his recent paper associates the onset of the second stage with the appearance of urinary changes. The amount of urine diminishes to 1000 c.c. or less in twenty-four hours, and it contains urobilin and albumen intermittently.

The liver begins to enlarge, until it is readily palpable three or four fingers' breadths below the costal margin in the right nipple line. It is smooth and painless. There is no ascites or jaundice.

Gastro-intestinal hemorrhages, particularly hæmatemesis, may occur before the onset of cirrhosis. Forty per cent. of the blood from the stomach reaches the portal system through the vasa brevia and splenic vein, and hæmatemesis is the mechanical result of congestion of the splenic vein. Other hemorrhages, subcutaneous, from the nose and gums, hæmaturia and melæna may also occur. Digestive disturbances and diarrhœa occasionally are observed.

The skin and conjunctiva may develop a grayish-yellow color, but actual pigmentation or jaundice does not occur in this stage.

With the approach of the third stage the liver diminishes in size. The second stage lasts from eighteen months to several years.

Third Stage.—The onset of the third stage is characterized by the development of ascites. This does not necessarily mean the presence of cirrhosis of the liver, since ascites frequently is seen before cirrhosis is demonstrable, as is shown by several operations and autopsy reports. Anæmia and enlargement of the spleen are the causative factors under these circumstances. The ascitic fluid has all the appearances of a transudate. It reforms rapidly after paracentesis.

The liver continues to diminish in size and well-marked cirrhosis develops. The gastro-intestinal hemorrhages are aggravated. The urine is still further diminished and contains urobilin and at times bilirubin. The skin develops a certain

amount of pigmentation in a few cases and late in the disease actual jaundice may occur.

Simonds in a critical study of fourty-seven cases of Banti's disease, from the literature, divides them into two distinct groups, each having definite pathological and clinical characteristics.

Group 1 consisted of thirty-three cases which were characterized pathologically by hyperplasia of all the connective-tissue elements of the spleen, and at times by marked congestion, by varicose veins in the lower end of the œsophagus and cardiac end of stomach, and frequently by cirrhotic changes in the liver and increase in the red bone marrow. The patients were twenty years or more of age when the symptoms began. The clinical characteristics of the cases in this group are splenomegaly, progressive anæmia, with a low color index, usually leukopenia, but never leukocytosis. These are constant findings and on them the diagnosis rests. Others, in order of frequency, are gastro-intestinal hemorrhage, ascites, diarrhœa, epistaxis, pigmentation of the skin and jaundice, which, if present, appears late in the disease.

Group 2 consisted of fourteen cases, which were characterized by diffuse proliferation of the endothelium of the spleen. The retroperitoneal lymph-nodes and liver sometimes show the same type of endothelial proliferation. The average age of onset of symptoms was fifteen years and there was an undoubted family tendency. Clinically this group is characterized by a more prolonged course than those cases included in group 1; there is splenomegaly; anæmia with a low color index; hemorrhages other than gastro-intestinal, such as nasal, oral, and subcutaneous; and absence of leukocytosis. Pigmentation and jaundice are less frequent.

Hill divides cases diagnosed splenic anæmia into a cachectic and a hæmolytic group. The *hæmolytic group* he considers examples of pernicious anæmia with enlarged spleen. Its characteristics are splenomegaly, a blood picture of the mild "pernicious" type, leukopenia, hæmatemesis and pigmentation. He thinks the occurrence of splenomegaly is due to splenophlebitis. The characteristics of the *cachectic group* are splenomegaly, anæmia of the secondary type with a persistently low color index, relative lymphocytosis, hemorrhages, transient ascites, slight en-

largement of the lymphatic nodes, and long duration without pigmentation.

Diagnosis.—The diagnosis of Banti's disease or splenic anæmia in the early stages, before the appearance of the anæmia, is impossible. Whatever the subsequent developments, at this stage the case is one of "idiopathic" splenomegaly. Even after the appearance of anæmia the differential diagnosis presents many difficulties. Fortunately the technique of splenectomy is so simple, and within recent years the indications have been so extended that a great many of the conditions that might be confused with splenic anæmia are now subjected to the same treatment, splenectomy.

The diagnosis, however, often may be made with reasonable certainty. The splenic enlargement first must be distinguished from tumors of other organs. This subject was considered at page 391. Certain other diseases must then be excluded. The most important of these are discussed below.

As **cirrhosis of the liver** is the terminal stage of Banti's disease, it is possible, in the later stages of the latter affection that doubt will arise as to the causation of the splenomegaly. If symptoms characteristic of hepatic cirrhosis, such as hæmatemesis, ascites, jaundice and decrease in the size of the liver, appear after the splenic enlargement has existed for some time, clinically the case is Banti's disease in the terminal stage; although it must be remembered that these symptoms do not necessarily mean that hepatic cirrhosis exists, as they may precede its development. Enlargement of the spleen occurring at the same time or after the changes in the liver and the onset of symptoms, indicates that the true condition is cirrhosis of the liver rather than Banti's disease. The history and physical signs may enable one to determine the form of cirrhosis which is present.

That this degree of simplicity in reaching a diagnosis is not always observed, is shown by the confusion that still exists in the classification of these cases. The three forms of hepatic cirrhosis most likely to be mistaken for splenic anæmia are the syphilitic, the alcoholic and the hypertrophic of Hanot.

In *Syphilitic Cirrhosis* of the liver the previous history, the Wasserman reaction, and the result of treatment are the main points on which reliance may be placed in reaching a diagnosis. Moreover, the enlargement of the liver due to syphilis is irregular.

Leukopenia is absent and in some cases there is a persistent but moderate leukocytosis. It must be remembered that a positive Wasserman reaction is not necessarily diagnostic, as there is no reason to suppose that a man with Banti's disease might not have had syphilis previously. Nor is the size of the spleen a sign to be depended upon, as this varies from moderate to great enlargement just as in Banti's disease. The anæmia of syphilitic cirrhosis may be very severe. Extreme anæmia of undertermined origin always suggests syphilis.

In *Alcoholic Cirrhosis*, the small liver, big spleen and anæmia suggest Banti's disease with recurring hemorrhages.

The age of onset, alcoholic history, early appearance of symptoms of cirrhosis and the late development of splenomegaly will serve for differentiation from all except the most rapidly developing cases of Banti's disease.

In the *Hypertrophic Cirrhosis of Hanot*, the great enlargement of the liver and the marked jaundice usually are sufficient to indicate that the enlargement of the spleen is secondary, even if the time of the occurrence of splenic enlargement in relation to hepatic enlargement is not known.

Hæmachromatosis.—This is a rare condition of biliary and splenic enlargement associated with hæmolysis of unknown origin. Pigmentation of the skin and internal organs results from the deposit of an iron-containing pigment. Hepatic and splenic enlargement occur synchronously.

Pernicious Anæmia.—Cases of pernicious anæmia with great enlargement of the spleen are not very uncommon and in certain stages readily may be mistaken for splenic anæmia. If the anæmia is extreme, the color index high, and if nucleated red blood cells and poikilocytes are present, the case is much more likely to be one of pernicious anæmia than of Banti's disease, even though the spleen is enlarged to the pelvic brim. Even if the changes in the blood characteristic of pernicious anæmia are absent, the presence of extreme anæmia is against Banti's disease unless there has been a recent severe hemorrhage to account for it. Even in the late stages of Banti's disease it is uncommon for the proportion of hæmoglobin to fall below 40 per cent. unless there has been recently a severe hemorrhage. Morris Lewis reported a case in which the hæmoglobin was 90 per cent. a short time before

the onset of hemorrhage and 18 per cent. a few days later. The history and blood picture usually serve to differentiate the two conditions, and confusion will be less apt to occur if it is borne in mind that a tremendously enlarged spleen does not necessarily rule out pernicious anæmia.

Hodgkin's Disease.—A splenic form of Hodgkin's disease, characterized by anæmia, enlarged spleen with lymph-adenomatous masses but without enlargement of the superficial lymph-nodes, has been described by some, but it is very questionable whether such a condition exists.

Malarial Hypertrophy.—Severe secondary anæmia with enlarged spleen as a sequel of malaria presents many of the characteristics of splenic anæmia. The previous history usually is definite enough to enable one to avoid a mistake in diagnosis. If repeated blood examination is negative, exploratory splenic puncture, as a rule, reveals the malarial parasite, but is open to the objection that it exposes the patient to the risk of severe hemorrhage. On account of the anæmia and the danger of rupture from slight degrees of traumatism splenectomy is becoming increasingly popular as a treatment for "ague cake" spleen.

Leukæmia is diagnosed by the blood picture. During remissions when the blood returns to normal, the diagnosis is difficult; but observation of the patient for a short time reveals the true condition.

Syphilis rarely is a cause of symptoms and physical signs resembling splenic anæmia. The Wassermann reaction and the result of treatment in doubtful cases are sufficient to establish the diagnosis. In adults this splenic enlargement usually is secondary to syphilitic affections of the liver, while in children syphilis causes primary splenomegaly. D'Arcy Power reported a successful case of splenectomy for simple hypertrophy, which turned out to be syphilitic in origin. Gumma of the spleen is of pathological rather than surgical interest.

Amyloid spleen always is secondary to causes, such as suppuration, tuberculosis, etc., which are easily demonstrable.

Tumors and **tuberculosis** of the spleen are discussed at page 403.

Splenomegaly in Childhood.—Except the hypertrophy occuring in *acute infectious diseases*, enlargement of the spleen in

infancy and early childhood is a condition of obscure ætiology. Congenital syphilis, rickets, disturbance of metabolism and intestinal disorders of doubtful classification are all considered ætiological factors in this group. It is very likely that some are cases of true splenic anæmia, but on account of the frequency of splenomegaly in childhood and the uncertainty of the blood picture, such a diagnosis always is doubtful.

Pseudoleukæmia infantium of von Jaksch, usually appearing in the second year of life, is characterized by very grave anæmia, by marked splenomegaly and usually by moderate enlargement of the liver. The red blood cells may be reduced to 1,000,000 and the hæmoglobin falls disproportionately, giving a low color index. The blood shows normoblasts, poikilocytosis, leukocytosis (15–20,000) and relative increase in lymphocytes. Two cases of splenectomy with recovery are reported, but medical treatment gives very good results, and even in severe cases iron, arsenic and mercury cause improvement and eventual cure in a large proportion of patients. One of these cases of splenectomy with recovery was in a child fifteen months old (Graff). The association of rickets with pseudoleukæmia is accidental, not causative. A history of congenital syphilis is present in about 50 per cent. of cases.

Cirrhosis of the liver with splenomegaly, ascites, and usually chronic jaundice, known as *splenomegalic cirrhosis*, occurs in children and presents no characteristic differences from the same condition in adults, except that there may be a family tendency. There are other forms of splenic enlargement of a family and hereditary type in children, one the so-called *congenital acholuric icterus* of Minkowski and the other a family type of *splenomegaly with dwarfing*, infantilism, etc. Surgical treatment has not as yet been undertaken for these cases.

Hæmolytic Splenomegaly.—Under this name Banti has quite recently described another disease first observed by him in 1903. It resembles both Banti's disease and hæmolytic jaundice, and is promptly cured by splenectomy. Some cases already reported as examples of Banti's disease he thinks really were instances of hæmolytic splenomegaly (Umber, Lintvarev). The fundamental symptoms are (1) splenomegaly, (2) anæmia, (3) jaundice. Patients come under treatment on account of the anæmia; the large spleen gives no trouble and may pass unnoticed. The anæmia

varies in intensity from time to time, over a period of years. During the worse phases the spleen grows larger, the urobilinuria increases, and the jaundice, if already present, deepens. During the better phases of the malady the red blood cells vary between three and four millions; in the bad phases they fall below one million. The color index usually is below 1; the hæmoglobin may fall even lower then 20 per cent. The red blood cells show anisocytosis, poikilocytosis, and polychromatophilia. There usually are some cells with basophilic granules, and there may be even normoblasts. The resistance of the R. B. C. is diminished: the initial hæmolysis may be 0.60 and the total hæmolysis 0.34. The white blood cells vary from 4500 to 11,500, usually being about normal. The differential count varies much from time to time. Myelocytes are seldom found, and only in the phases of intense anæmia. Jaundice may not develop for many years; always is slight, but grows worse during the phases of severe anæmia. The fæces always are well colored; urobilin always is present in the urine. The liver often is enlarged, especially during the phases of severe anæmia. Death occurs in from six to twelve years unless splenectomy is done; and this is the only measure which has any effect. In three cases thus treated, recovery ensued in all.

Prognosis and Treatment.—Medical treatment is absolutely worthless as regards the cure or arrest of the disease. With the most skillful administration of drugs or other therapeutic agents, such as the X-rays, the course of the disease is steadily toward a fatal termination. Splenectomy is the only form of treatment that offers any hope of permanent cure. As the operations and reported cases multiply and as the after-results are studied, it is becoming more and more evident that surgery effects an absolute cure in the first stage of the disease in every patient who makes an operative recovery. In the second stage the primary mortality is higher and the percentage of permanent cures is less. In the third stage the disease is too advanced to expect any good results from splenectomy, but Talma's operation may be undertaken at this time with moderate hope of success in removal of the symptoms of cirrhosis. One point should be thoroughly understood: ascites and slight or transient jaundice occurring in the course of Banti's disease do not absolutely contraindicate splenectomy, because they may occur before cirrhosis of the liver is present. The

latter is the only contraindication to splenectomy, and it is not an absolute contraindication. Banti reported ten operations on his own patients and twenty collected operations with the following results:

	Banti's Cases.		Collected Cases.	
	Operations.	Cures.	Operations.	Cures.
First period	2	2	2	1
Second period	6	5	16	8
Third period	2	0	2	1

From January 1, 1908, to January 1, 1912 there were reported thirty-seven operations for Banti's disease with nine operative deaths, a mortality of 24.3 per cent.

Splenectomy is recognized as the only rational treatment for Banti's disease, and better results are to be expected in the future with earlier resort to operation. Splenectomy should be undertaken as soon as the diagnosis is made, and the knowledge that the disease can be cured by operation should bring home to every physician the importance of early diagnosis.

The success of splenectomy seems to indicate that the spleen is closely associated with the causation of the changes characteristic of the second and third stages.

Splenocleisis.—Under this name Schiassi, according to Citernesi and Ficai described an operation for Banti's disease in which the enlarged spleen is not removed, but is packed all around with iodoform gauze. This is to be allowed to remain for from six to ten days, by which time sufficient adhesions will have formed to ensure reduction in size of the spleen by their ultimate contraction. The large veins formed in this way are supposed to divert the blood from the spleen into the abdominal wall. Mugnai adopted this method in one case, but the patient died from internal hemorrhage the next day.

REFERENCES.

Bovaird: Am. J. Med. Sc., 1900, ii, 377.
Banti: Dell'Anemia Splenica, Firenze, 1882.
Banti: Folia Hæmatolog., 1910, x, 33.
Banti: Semaine Méd., 1912, xxxii, 265 (Hæmolytic Splenomegaly).

Citernesi and Ficai: Arch. de Méd. Exper. et d'Anat. Path., 1910, xxii, 900.
Graff: Verhandl. d. Deutsch Ges. f. Chir., 1908, xxxvii, 248.
Hill: St. Barth. Hosp. Repts., 1909, xlv, 43.
Lewis, M. J.: Amer. J. Med. Sc., 1908, ii, 157.
Oettinger and Fiessinger: Rev. de Méd., 1907, xxvii, 1109 (Splenophlebitis).
Osler: Brit. Med. J. 1908, ii, 1151.
Osler: Amer. Jour. Med. Sc., 1900, i, 54; 1902, cxxiv, 751.
Power, D'A.: St. Barth. Hosp. Repts., 1908, xliv, 95.
Risel: Beitr. z. path. Anat., 1909, v, 241.
Rolleston: Clin. Jour., London, 1901–02, xix, 401; 1908–09, xxxiii, 1.
Schiassi: Anemia Splenica, Firenze, 1906, quoted by Citernesi and Ficai, l. c.
Severino: Anemia Splenica e Morbi Affini. Napoli, 1911.
Simonds: Jour. of Infect. Dis., 1908, v, 23.
Sutherland and Burghard: Lancet, 1910, ii, 1819.
Torrance: Mobile Med. and Surg. Jour., 1908, xiii, 125.

Injuries of the Spleen.

These may be divided into cases of *subcutaneous rupture*, almost always due to indirect violence, or to blunt force if the injury is direct, and *open wounds*, especially gunshot and stab wounds.

Disease of the spleen associated with enlargement frequently is a predisposing cause.

Subcutaneous Rupture.—If the spleen is normal a very great degree of trauma is necessary to rupture it. Being run over by various vehicles, falls from a height, kicks and crushes are the usual causes of subcutaneous rupture. Violence may be applied directly to the spleen beneath the costal arch or indirectly through the ribs. In the latter case, fracture is not a necessary concomitant as the spleen often has been severely injured although the ribs remained intact. Rupture from indirect violence has also followed blows on the right side and even falls on the feet. If the spleen is enlarged it is easily ruptured by an insignificant degree of traumatism, such as tightening a belt, coughing, lifting, etc. This violence may be so slight that the case appears almost one of idiopathic rupture, but in the majority of cases a history of some slight injury is obtainable. There are, however, a certain number of cases in which rupture has occurred when the patient was lying quietly in bed. These are examples of true spontaneous rupture,

although they are the result of advanced disease of the spleen, such as tuberculosis, or more particularly malaria. In the tropics rupture of a malarial spleen is a common cause of sudden death. Labor or eclampsia may be the exciting cause of rupture if the spleen is enlarged. Bryan collected and reported thirty-five cases of spontaneous rupture of the spleen occurring during the course of typhoid fever.

Pathology.—Laceration of the splenic substance varies from a slight subcapsular rupture of the pulp to complete disorganization. The spleen may be divided into two or more pieces or it may be completely torn away from its vessels. Hemorrhage is the most important factor. If the capsule is torn the blood is poured out into the general peritoneal cavity and the spleen shows only the rent in its substance. If the capsule is untorn (subcapsular rupture) the blood collects beneath it and forms a hæmatoma. As the pressure increases, it may cause cessation of the bleeding or rupture of the capsule. In the former case a hemorrhagic cyst is formed, in the latter, a frank rupture. If clotting occurs and bleeding stops, the future condition of the spleen depends on the degree of injury. If any of the main vessels are involved necrosis will follow. If only the splenic pulp is torn, the wound may heal, or, as is more likely, softening of the clot will lead to secondary hemorrhage in a few days.

Site of Rupture.—If extensive injuries involving a large portion of the splenic substance are excluded, the site of rupture occurs five to six times more frequently on the internal surface than on all the rest of the surfaces collectively. Frequently the laceration extends from the inner to one or more of the other surfaces. If the rupture is limited to the internal surface immediately behind the hilum the bleeding may be confined to the lesser peritoneal cavity.

Rupture of Enlarged Spleen.—The same degrees of injury occur as in the normal spleen, and the difference in morbid anatomy is that the spleen shows the changes characteristic of the pathological process to which it is subject.

Injury to other Organs.—On account of its position, rupture of the spleen frequently is accompanied by injury to one or more of the surrounding organs, stomach, liver, intestine, kidney, pancreas and suprarenal gland. These injuries vary in severity as do

those in the spleen. Fracture of the ribs and bruises on the surface of the body may indicate the point of application of the causative force, but very often there is no external evidence of injury.

Penetrating Wounds.—Stab wounds vary in depth and breadth according to the size of instrument and its depth of penetration. Gunshot wounds may cause great disorganization of the spleen or only small wounds of entrance and exit.

Injury to the surrounding structures is even more frequent than in subcutaneous rupture, particularly in gunshot wounds.

Thévenot collected eighty-one cases of gunshot and stab wounds of the spleen, and twenty-four of them were complicated by wounds of one or more of the surrounding viscera. The viscera injured were the

Stomach,	in 14 instances.
Liver,	in 5 instances.
Kidney,	in 4 instances.
Intestine,	in 5 instances.
Omentum,	in 2 instances.
Lungs,	in 2 instances.

In several cases more than one other organ was injured. In addition, the diaphragm and pleura are involved more frequently than any of the viscera.

Symptoms.—As the symptoms from subcutaneous and penetrating wounds do not differ very much, they may be considered together.

They vary from sudden and immediate death, to the gradual or sudden onset of symptoms seventy-two hours or more after the injury. Sudden death, usually due to shock and profuse internal hemorrhage, may not be attributable solely to the splenic injury but may be due chiefly to complicating lesions. When the onset of symptoms is delayed, it is probable that the original injury produced a subcapsular hæmatoma which subsequently ruptured; or a primary complete but small rupture may have been sealed temporarily by clotting, and the late symptoms may be caused by dislodgment of this clot with secondary hemorrhage. In either case the symptoms, although appearing late, are the same as those that occur immediately after the injury.

Sudden, severe pain occurs in the left hypochondrium accompanied by a sense of tearing. The symptoms of shock due to internal hemorrhage appear with a varying degree of rapidity. Subsequently the pain in the left hyponchondrium varies in severity from a more or less continuous dull ache, with acute exacerbations, to pain so insignificant as to be almost negligible. Levy called attention to the value of sharp pain in the left shoulder region as a sign of rupture of the spleen. Breathing and moving aggravate whatever pain is present. The time of appearance of the signs and symptoms of shock depends on the rapidity of the bleeding; very often a large amount of blood escapes slowly into the abdomen before appreciable symptoms appear. The two most important signs are increasing pallor and rise in the pulse rate with diminution of its volume. Subsequently the patient exhibits a rapid, running pulse, restlessness, vomiting, air hunger, and other signs of severe internal hemorrhage. Abdominal distention with rigidity more or less localized to the left upper quadrant may be present, and with these signs there may be increase in the area of splenic dullness and dullness in the left flank instead of the normal resonance. Dullness in the flank when present often is not a shifting dullness. Frequently all these signs are absent although there is an extensive laceration. Tenderness over the spleen usually is demonstrable either from in front or behind.

Rupture in the course of typhoid fever as a rule is mistaken for intestinal perforation. The rupture is manifested by pain beginning in the splenic region, by marked abdominal rigidity, followed by distention, with small rapid pulse, and extreme toxæmia. A drop of the temperature to subnormal may occur at the time of rupture, as in cases of intestinal perforation, but, as also in these cases, the temperature rises as peritonitis develops.

Rupture of a malarial spleen causes the rapid appearance of shock from internal hemorrhage, and in most cases this is quickly fatal.

Diagnosis.—History of an injury, pain in the left hypochondrium, symptoms of internal hemorrhage, dullness in the flank and localized rigidity, when present all together leave very little doubt of the diagnosis. Injury to the kidney is ruled out by the absence of blood in the urine. Intestinal injury gives rise to earlier

and more marked abdominal rigidity, which usually is more generalized; and the symptoms of internal hemorrhage are likely to be less severe unless the mesenteric vessels are torn across. The site of the blow usually excludes liver injury; but in certain cases, and particularly those where the splenic rupture is due to indirect violence, differentiation is practically impossible. Rupture of the pancreas rarely occurs without injury to the surrounding organs and it frequently is involved with the spleen. The stomach and gall-bladder rarely are injured except in the case of panetrating wounds. Stomach injury may lead to hæmatemesis; otherwise the symptoms are those of intestinal rupture. Laceration of the gall-bladder resembles injury to the liver except that the symptoms of hæmorrhage are not marked.

As the treatment for all these conditions is the same, laparotomy, time ought not to be wasted in attempting to differentiate, but operation should be undertaken as soon as it is evident that there has been visceral injury. If the surgeon waits for shock to subside few patients will be saved from death.

Prognosis and Treatment.—The treatment of splenic injuries is immediate laparotomy. This is followed by packing of the wound, by its suture, or by splenectomy, according to the extent of the injury to the spleen. The mortality of cases in which no operation is done is 95 per cent. or more. The operative mortality is about 33 per cent.; this, however, does not represent the full mortality of rupture of the spleen, as many patients die too soon to allow time for operation. There are several reported recoveries from injuries that could not be distinguished clinically from rupture, but there must always be a large element of doubt in the diagnosis when the patient recovers without operation.

Operative Treatment.—The incision should be made in the epigastrium just to the left of the middle line, so as to permit thorough exploration and treatment of concomitant injuries.

The operation selected depends upon the position and extent of the injury. Splenectomy is more often undertaken than all other operations combined, but repair of the wound, plugging with gauze, and suture of the omentum to the wound have all been recommended and performed with success.

Splenectomy is done most frequently because it is the most certain way to stop the bleeding; because the operation is easily

performed in the majority of cases; and, more particularly, because the greater number of splenic injuries are of such a nature that they cannot be treated in any other manner with a reasonable hope of success. There are reported 227 cases of splenectomy for injury with 155 recoveries and 72 deaths, a mortality of 31 per cent. In a large proportion of the fatal cases there were other visceral injuries.

Suture.—Even when the injury is small it is very difficult to control hemorrhage with sutures. In a report of 134 cases of rupture of the spleen, Lotsch gives the following results: splenectomy, 118 cases with fifty-two deaths; splenorrhaphy, eight cases with three deaths; tamponade eight cases with two deaths. Kirschner described a method of encapsulating the wound by sewing the omentum to the edges. He operated on four cases with one death, and in this case operation was refused until the patient was almost moribund.

Tamponade.—The advantage of plugging with gauze is that the spleen is preserved. This undoubtedly furnishes a strong argument in its favor, but the control of hemorrhage is uncertain in all but the most favorable cases. Tamponade is particularly indicated in those cases where the presence of perisplenic adhesions renders splenectomy difficult or impossible. It may be advisable to soak the gauze in adrenalin solution.

Temporary Clamping of Vessels.—Sheldon, as a result of experimental work on dogs, advises the use of rubber-covered clamps applied to the pedicle for four hours. They should then be loosened, and if hemorrhage does not recur may be removed. They may be reapplied for another two hours if hemorrhage persists. In his experiments hemorrhage has not recurred when the clamp was removed in four hours. The splenic wound is not treated. By this means the vessels empty themselves without clotting. If the wound is packed the clot extends back through the vessels to the clamp and necrosis might result.

REFERENCES.

Bryan: Annals of Surg., 1909, l, 856.
Elliot: Trans. Amer. Surg. Assoc., 1907, xxv, 336.
Kirschner: Amer. J. Obst., 1909, lix, 431.
Levy: N. Y. Med. J., 1908, lxxviii, 693.

Lorenz: Zentr. f. Chir., 1910, xxxvii, 1577.
Leitner: N. Y. State J. of Med., 1910, x, 256.
Lotsch: Deutsch. Zeit. f. Chir., 1908, xciii, 90.
LeMoine et Leveque: Bull. Soc. Anat., Paris, 1907, lxxxii, 511.
Lithgow: Jour. Royal Army Med. Corps, 1908, xi, 82.
Nötzel: Beitr. z. klin. Chir., 1908–09, lxi, 215.
Sheldon: Amer. J. Med. Sc., 1910, cxxxix, 581.
Ross, G. G.: Annals of Surgery, 1908, xlviii, 66.
Thévenot: Prov. Méd., 1910, xxi, 245.

Operations on the Spleen.

The following list comprises the conditions for which **splenectomy** may be advisable or necessary.

Hypertrophy—idiopathic and malarial.
Wandering spleen.
Twisted pedicle.
Splenic anæmia (Banti's disease).
Hæmolytic splenomegaly.
Cysts, hydatid and non-parasitic.
New growths.
Tuberculosis.
Pseudoleukæmia.
Abscess.
Wounds and injuries.

Splenectomy for Idiopathic Hypertrophy.—The number of cases classed as idiopathic hypertrophy is gradually diminishing, because of more careful classification. A great many supposedly simple enlargements eventually prove to have been cases of Banti's disease in the first stage. The difficulty of diagnosis is very great in the tropics where there are so many splenomegalies of obscure origin.

Splenectomy is indicated in cases of simple enlargement because of the danger of rupture from slight injuries. In addition, there is sometimes considerable discomfort from the presence of the tumor, and its weight and mobility often are responsible for various gastro-intestinal disturbances.

RESULTS OF OPERATION.

	Recovered.	Died.	Total.	Percentage Mortality.
Before 1900	20	13	33	39.4
1900–1908	33	8	41	19.5
1908–1912	7	0	7	0.0
Total	60	21	81	25.9

Splenectomy for Malarial Hypertrophy.—Spontaneous rupture and rupture from slight degrees of traumatism are very frequent in malarial hypertrophy. On this account and also because patients invariably show marked improvement after operation, splenectomy is indicated in certain cases, particularly those showing persistent anæmia.

RESULTS OF OPERATION.

	Recovered.	Died.	Total.	Percentage Mortality.
Before 1900	58	30	88	34.1
1900–1908	53	8	61	13.1
1908–1912	13	3	16	18.75
Total	124	41	165	24.8

Splenectomy for Wandering Spleen.—Simple mobility of a normal sized spleen, without symptoms, is not an indication for surgical treatment. These cases usually are discovered accidentally. If the spleen is enlarged, the likelihood of rupture is greatly increased by its mobility, and twist of the pedicle is a constant danger. A movable spleen is prone to attacks of perisplenitis and the formation of adhesions to various structures. Interference with the functions of other organs and symptoms from the contraction of adhesions result. Dragging of the spleen on its pedicle also causes various more or less indefinite abdominal symptoms. Splenectomy therefore is indicated in all cases of movable spleen except those unaccompanied by any signs or symptoms referable to its mobility. Very often a correct diagnosis is not made until the abdomen is opened, the misplaced spleen simulating some other abdominal tumor. Particularly is this true of a spleen adherent in the pelvis or in either iliac fossa.

RESULTS OF OPERATION.

NON-MALARIAL CASES.

	Recovered.	Died.	Total.	Percentage Mortality.
Before 1900	41	5	46	10.9
1900–1908	13	1	14	7.1
1908–1912	4	0	4	0.0
Total	58	6	64	9.4

MALARIAL CASES.

Before 1900	25	1	26	4
1900–1908	14	0	14	0
1908–1912	2	0	2	0
Total	41	1	42	2.4

Splenectomy for Twist of the Pedicle.—This is an absolute indication for splenectomy, although there are several cases reported where the pedicle was untwisted and the operation successfully completed by splenopexy. The latter seldom is advisable, because of the condition of the spleen. It usually is soft and friable, and may show beginning necrosis as the result of interference with its blood supply. Thrombosis of the vessels and infarcts often are present. For these reasons there is frequently considerable damage done to the spleen during the manipulations necessary to free it from adhesions and untwist the pedicle. Therefore splenectomy is less dangerous than splenopexy.

If the twists in the pedicle are so loose that they do not interfere with the blood supply, it is clinically a case of movable spleen.

RESULTS OF OPERATION.

NON-MALARIAL CASES.

	Recovered.	Died.	Total.	Percentage Mortality.
Before 1900	8	8	16	50
1900–1908	11	0	11	0
1908–1912	5	0	5	0
Total	24	8	32	25

Malarial Cases.				
Before 1900	3	2	5	40
1900–1908	7	0	7	0
1908–1912	3	0	3	0
Total	13	2	15	13.3

Splenectomy for Splenic Anæmia.—Splenectomy is indicated as soon as the diagnosis is made, unless it is certain that advanced cirrhosis of the liver is present.

In the stage of simple enlargement the indications for splenectomy are the same as in idiopathic hypertrophy. The advent of anæmia establishes the diagnosis and there is no longer any reason to delay operation, as it offers the only chance of cure. After the onset of cirrhosis of the liver, splenectomy is powerless to cure the condition and the operative mortality is high; but there are a certain number of cases on record in which splenectomy, sometimes combined with epiplopexy, arrested the progress of the disease for some years, if not permanently. These were cases in which definite granular changes in the liver were present. Several patients with advanced cirrhosis recovered from the operation, but died within a few weeks from continuation of the disease. But as no other treatment is of any avail and as even in this advanced stage there is a fair chance of amelioration of the symptoms by splenectomy and epiplopexy, the patient should be given a chance if there is any reasonable hope of operative recovery. As pointed out at page 413, ascites may result from the splenomegaly and the anæmia, and does not necessarily mean that cirrhosis has started. It is difficult to differentiate these two causes without opening the abdomen, as the other common sign of cirrhosis, hæmatemesis, is an early symptom of splenic anæmia, occurring before there are any changes in the liver.

Hill says that splenectomy does not cure the hæmolytic form of the disease (see page 414), but delays the appearance of the common causes of death, hæmatemesis, cirrhosis and ascites. On account of the favorable results obtained in this class, which he believes to be cases of pernicious anæmia with splenomegaly, he advocates splenectomy as a treatment for pernicious anæmia. He considers that cachectic cases are permanently cured by splenectomy. Banti strongly urges splenectomy as the only cure

for the disease he has described as hæmolytic splenomegaly (p. 418).

It is still very difficult to reach satisfactory conclusions on the subject of permanent results, as the case reports are not always definite in relation to the stage of the disease, and as the after history of many of the patients is not known for any considerable period. But there can be no doubt that removal of the spleen is followed by marvellous improvement which persists for years if the operation is performed in the first or second stage of the disease.

RESULTS OF OPERATION.

	Recovered.	Died.	Total.	Percentage Mortality.
Before 1900	12	5	17	29.4
1900–1908	37	7	44	15.9
1908–1912	28	9	37	24.3
Total	77	21	98	21.4

Splenectomy for Hydatid and Non-parasitic Cysts.—Splenectomy is indicated in the majority of cases of cyst of the spleen, but occasionally enucleation and repair of the raw surfaces is possible. If the adhesions are so dense that splenectomy is prevented formolization, as in hydatid cysts of the liver (page 199) should be done. Marsupialization consists in cutting away a portion of the cyst, removing the inner cyst wall and suturing the edges of the cavity to the abdominal wound, through which it is packed until it closes by granulation and contraction. Splenectomy is the operation of choice.

RESULTS OF SPLENECTOMY.

Non-parasitic Cysts.

	Recovered.	Died.	Total.	Percentage Mortality.
Before 1908	19	0	19	0.0
1908–1912	5	1	6	16.7
Total	24	1	25	4

Hydatid Cysts.

	Recovered.	Died.	Total.	Percentage Mortality.
Before 1900	11	4	15	26.7
1900–1908	8	0	8	0.0
1908–1912	4	2	6	33.3
Total	23	6	29	20.3

Splenectomy for New Growths.—Splenectomy has been performed for sarcoma, secondary carcinoma, angeioma, myxofibrolipoma, and for a large intraperitoneal lipoma with involvement of the spleen. There is nothing characteristic in these conditions upon which to base a diagnosis, except that in cancerous angeioma pulsations may be felt. All these tumors are surgical curiosities, with the possible exception of sarcoma, for which fifteen splenectomies are reported with twelve recoveries and three deaths. One of these cases is tabulated under rupture of the spleen. One splenectomy for sarcoma of the spleen, with operative recovery, has been done by the senior author at the German Hospital.

Splenectomy for Abscess.—Splenectomy is indicated if the spleen is free from adhesions and can be removed without much injury to surrounding organs. As a rule, by the time the diagnosis is made the spleen is surrounded by adhesions and excision is difficult or impossible. In these cases incision and drainage is the only available treatment. Johnston's table contains nine cases of splenectomy for abscess with one death. In some of them splenectomy was undertaken as a secondary operation either because of recurrence or to cure a persistent sinus. As stated at page 408, no case of splenectomy for abscess appears to have been reported since 1908.

Splenectomy for Tuberculosis.—The diagnosis of tuberculosis of the spleen is one that cannot be made with certainty. In the reported cases splenectomy has been undertaken for rupture or for constitutional symptoms, particularly anæmia accompanied by splenomegaly. Fifteen splenectomies with three deaths are reported.

Splenectomy for Pseudoleukæmia.—The conditions to which this name has been and is applied are more or less obscure.

Pseudoleukæmia infantium of von Jaksch is briefly described at page 418. Johnston mentions five splenectomies for pseudoleukæmia from 1900 to 1908, and comments on the diagnosis as follows: One probably was malaria, one probably splenic anæmia, two were pseudoleukæmia infantium, and one was of unknown origin. Since 1908 there have been two splenectomies for pseudoleukæmia reported, one by Graff in a child fifteen months old, the other by Orsos in a woman forty-eight years old. Both recovered.

Under these circumstances it is extremely difficult to make any rules of treatment that will apply to the individual case. Each one must be treated on its merits, and splenectomy undertaken if medical treatment fails.

Splenectomy for Wounds and Injuries.—The treatment of injuries and the indications for splenectomy have been described (page 425).

RESULTS OF OPERATION.

	Recovered.	Died.	Total.	Percentage Mortality.
Before 1900	20	17	37	45.9
1900–1908	79	34	113	30.1
1908–1912	56	21	77	27.3
Total	155	72	227	31.7

Splenectomy for Leukæmia.—Splenectomy is absolutely contraindicated in any form of leukæmia. The primary mortality is 88 per cent. and the progress of the disease is not delayed by the operation.

Splenectomy for Aneurism of the Splenic Artery.—This was done successfully by Winckler, who refers to one similar operation by Selten, and says that Ponfick had found an aneurism of the splenic artery four times at autopsy. Villard and Murard attempted splenectomy in one case, but adhesions rendered this impossible. The pedicle of the aneurismal tumor had to be clamped to check bleeding, and the clamps were left in place, as ligation was impossible. The patient died in ten days from secondary hemorrhage. In both Winckler's and Villard's patients the chief symptoms were recurring attacks of pain of some years duration. In

both the operation was exploratory, a correct diagnosis not having been made before opening the abdomen.

REFERENCES.

Villard and Murard: Arch. Gén. de Chir., 1912, vi, 749.
Winckler: Zentr. f. Chir., 1905, xxxii, 257.

Prognosis after Splenectomy.

In summarizing the results of splenectomy from 1900 to 1907, inclusive, Johnston gives the following table.

Total	355 cases	66 deaths	18.5 per cent. mortality.
Excluding injuries	242 cases	32 deaths	13.2 per cent. mortality.
Excluding leukæmia	235 cases	27 deaths	11.5 per cent. mortality.

The reason for excluding injuries and leukæmia in attempting to arrive at some conclusion of the dangers of splenectomy, is obvious. In rupture of the spleen the prognosis is complicated by other injuries and the patient frequently is almost moribund when operated upon; in other words, the injury and not the operation is the cause of death. The mortality in leukæmia makes it now a disease in which splenectomy is contraindicated, and it should be excluded from any list in which the probable future mortality is forecasted.

The following table is a summary of the cases from 1908 to 1911, inclusive.

Total	176 cases	37 deaths	21.02 per cent. mortality.
Excluding injuries	99 cases	16 deaths[1]	16.2 per cent. mortality.

Male	77
Female	64
Not mentioned	35
	176

The results from 1908 to 1912 do not compare favorably with those shown in Johnston's table. The explanation is hard to find unless it is that the operation is undertaken in more advanced cases than formerly. On the other hand a great many of the cases reported since 1908 were operated upon some years before. Although the statistics of reported cases are the only data upon which

[1] Banti's disease accounts for nine of these sixteen deaths.

to depend in making a prognosis, two factors militate against their value. There is always a greater tendency to report successful than unsuccessful cases, but, at the same time, the greater number of operators have performed very few operations, many only one, and the mortality is bound to be influenced by this lack of experience. Giffin states that among twenty-seven splenectomies done by Mayo there were only two operative deaths (7.4 per cent).

The dangers of splenectomy are shock, hemorrhage and the subsequent development of peritonitis. On account of the necessity for hurry in a large number of cases, subphrenic abscess, wound infection, and incisional hernia are camparatively frequent sequels. Pneumonia and pleurisy are not more frequent than after other upper abdominal operations, if traumatic cases are excepted. In the latter the proportion of patients showing postoperative pneumonia and pleurisy is greater, probably owing to concomitant thoracic injuries. Delirium, high fever and other untoward results practically always are due to sepsis.

REFERENCES.

Splenectomy.

(All cases of Splenectomy previous to 1908 have been tabulated by G. B. Johnston: Annals of Surgery, 1908, xlviii, 50. The following references are to cases of splenectomy reported since January 1, 1908. They have been collected for us by Dr. J. R. Freeland, formerly resident physician in the German Hospital, Philadelphia.)

Albrecht, P.: Mitt. d. Gesellsch f. inn. Med. und Kinderh. in Wien, 1908, vii, 189. Tuberculosis.

Alivisatos, N.: (Alibizatos), Bull. Soc. Chir., Paris, 1909, xxxv, 85. Traumatic rupture of spleen and stomach with multiple ruptures of the omentum in malaria.

Banti: Translated by Rober and Tissot. Folia Hæmatolog., 1910, x, 33. "Ueber Morbus Banti."

Baudet, R.: Bull. et mém. Soc. de Chir. de Par., 1908, n. s. xxxiv, 1209. "Contusion du rebord thoracique gauche; rupture complète de la rate; splenectomie; guérison." (Rapport par Mauclaire.)

Begouin: Gaz. hebd. d. sc. méd. de Bordeaux, 1908, xxix, 140. "Kyste hydatique de la rate datant de 44 ans; splenectomie; guérison."

Biondi, D.: La Riforma Medica, 1911, xxvii 365. "Un caso di echinococcus della milza."

Blecher: Muenchener med. Wchnscht., 1911, lxiii, 1312. "Subkutan traumatische Milz-Zerreisung bei Morbus Banti; Splenectomie; Heilung."

Bogart, A. H.: Annals of Surgery, 1908, xlvii, 37. "Splenectomy for lacerated spleen after blood transfusion."

Bragsitter, C. M.: Charitè Ann., Berlin, 1909, xxxiii, 494. "Splenektomie und sutkutane Milzruptur; Historiches, Kasuistisches u. Kritisches."

Burnier, R.: Bull et. mém. Soc. d'anat. de Par., 1910, lxxxv, 319. "Rupture traumatique la rate; Splenectomie."

Bush, C.: Jour. Am. Med. Assoc., 1910, liv, 453. Primary Sarcoma of the Spleen.

Corner: Med. Press and Circ., 1909, n. s., lxxxvii, 426. Ruptured Spleen.

Darling: Medical Record, Jan. 21, 1911, p. 110. Non-parasitic cyst of the spleen. Splenectomy.

Davis, A. B.: Bull. Lying-in-Hospital, N. Y., 1908, v, 24. Splenectomy for enlarged wandering spleen complicating pregnancy.

DeMarchis, F.: Gazz. d. Osp. Milano, 1908, xxix, 301. "Sulle cisti emboliche della milza."

Dartschlander: Deutsche med. Woch., 1908, xxxiv, 174. Milztumor.

Diffenbaugh, W. B.: Med. Herald, 1910, n. s., xxix, 56. Splenectomy for traumatic rupture of spleen, with report of a case.

Elliot, E., Jr.: Annals of Surgery, 1909, xlix, 711. Ruptured spleen; splenectomy.

Fagge, C. H., and Maun, H. C.: Guy's Hospital Rep., 1908, lxii, 135. A case of traumatic rupture of the normal spleen, splenectomy.

Fisher, W. E.: Proc. Roy. Soc. Med., 1908–09, ii, Clin. Section 128. Splenic infarction. Splenectomy.

Fiske, E. H.: Annals of Surgery, 1908, xlvii, 38. Splenectomy for gunshot wound of spleen.

Fowler, R. H.: Surg., Gynec. and Obst., 1910, xi, 133. Non-parasitic cyst of spleen.

Gemmell, J. E.: Jour. Obst. and Gyn. Brit. Emp., 1908, xiv, 237. Two cases of enlarged wandering spleen simulating pelvic tumor.

Gerrard, P. W.: Jour. Trop. Med., 1908, xi, 199. Traumatic rupture of the spleen. Splenectomy. Recovery.

Gervino, A.: Boll. d. r. Accad. med. di Genova, 1909, xxiv, 166. Le crasi sanguigne di uno splenectomizzato.

Graff: Verhandl d. deutsch Gesellsch f. Chir., 1908, xxxvii, 248. Ueber Milzextirpation bei anæmia pseudoleukæmica infantum.

Graham, G., and Chambers H. W.: West Lond. Med. Jour., 1910, xv, 46; 67. A case of ruptured spleen.

Guibè, M.: Trois cas de splenectomie pour coups de feu de la rate. Rapport du M. Auvray.

Hall, F. deH, and Spencer, W. G.: Proc. Roy. Soc. Med., 1908–09, ii, Clin Sect. 238. Splenectomy for splenic anæmia.

Harris, W. L.: Providence Med. Jour., 1908, ix, 7. Two cases of splenectomy.

Hauptmann, F. J.: Med. Klin. 1910, vi, 265. Ein Fall von primaren Milzsarkom.

Hoffmann, A.: Beitr. z. klin. Chir. 1909, lxiii, 778. Beitr. zu den subkutanen Milzruptur.

Holt, M. P.: Jour. Royal Army Med. Corps, 1909, xiii, 189. Successful splenectomy for rupture of large malarial spleen in European boy.

Hunt, P.: Jour. Amer. Med. Assoc., July 8, 1911. Report of Arkansas Med. Soc. Splenectomy for gunshot wound.

Kammerer, F.: Annals of Surgery, 1909, l, 484. Splenectomy for splenic anæmia.

Kappis: Klin. Ther. Wchnschr., 1911, xviii, 36. Milz Exstirpation.

Kirschner, W. C. G.: Amer. Jour. Obstet., 1909, lix, 431. Surgery of spleen, with especial reference to trauma, a method of encapsulation and report of cases.

Kummer: Rev. Méd. de la Suisse Rom., 1908, xxviii, 72. Splenectomie pour lymphosarcome de la rate.

Küttner, H.: Beitr. z. klin. Chir. 1907, liv., 405. Ueber Sequestrierende Milzabscesse.

Lecène, P.: Bull. et mém Soc. d'anat de Par., 1908, lxxxiii, 318. Rupture traumatique de la rate; splenectomie; guérison.

Levy, J. H.: N. Y. Med. Jour., 1908, lxxxviii, 693. A case of splenic anæmia. Splenectomy. Blood changes.

Lorenz, L.: Zentralblatt für Chirurgie, 1910, xxxvii, 1577. Zur Diagnose der Milzverletzungen.

Lotsch: Deutsche Ztschr. f. Chir., 1908, xciii, 90. Die subkutane milzzerreissung und ihre Behandlung.

Lukis, T. S.: Lancet, 1909, i, 1747. Rupture of the spleen; removal; recovery.

MacDonald, G. C.: Pacific Med. Journ., 1909, lii, 653. Splenectomy.

MacDonald, I. and Mackay, W. A.: Lancet, 1909, ii, 917. A case of acute torsion of a wandering spleen; splenectomy; recovery.

McGannon, M. C.: Trans. South. Surg. and Gynec. Assoc., 1908, xxi, 304. Splenectomy for splenomegaly of doubtful classification.

McCoy, J. C.: Annals of Surg., Nov., 1911. Splenectomy for rupture of the spleen.

MacLaren, A.: Annals of Surgery, 1910, li, 834. Wandering spleen, report of a case with twisted pedicle, etc.

Mallett, G. N.: Med. Rev. of Rev., 1910, xvi, 321. A case of traumatic rupture of the spleen; splenectomy; recovery.

Martin, A.: Rev. de gynec. et de chir. abd., 1909, xii, 67. Angiome de la rate.

Martin, A.: Arch. gén. de chir., 1908, ii, 20. Kystes hydatiques de la rate.

Mauclaire: Bull. et mém. Soc. de Chir. de Paris, 1910, n. s., xxxvi, 594. Rupture traumatique de la rate.

Mayo, W. J.: Jour. Amer. Med. Assoc., 1910, liv, 14. The principles underlying the surgery of the spleen, with a report of ten splenectomies. (For a summary of twenty-seven splenectomies by Mayo, see Giffin: Amer. Jour. Med. Sc., 1913, i, 781.)

Meyers, J.: Jour. Amer. Med. Assoc., 1909, lii, 1231. A study of the blood after splenectomy following trauma.

Michon: Bull. et mém Soc. de Chir. de Paris, 1909, n. s., xxxv, 822. Kyste hydat. de la rate.

Milward, F. V. P.: Rep. Soc. Study Dis. Child. 1908, viii, 406. A case of ruptured spleen in a child aged eighteen months; splenectomy; recovery.

Momm: Deutsche med. Wchnschrt., April 28, 1910.

Muhsam, R.: Deutsche med. Wchnschrt., 1910, xxxvi, 800. Milzruptur, Milzexstirpation, Heilung.

Niblock: Indian Med. Gazette, 1911, xlvi, 136. Rupture of spleen, excision; recovery.

Northrop, H. L.: Hahnemann Monthly, 1910, xlv, 109. Primary Splenomegaly; splenectomy and recovery.

Ochsner, A. J.: Illinois Med. Jour., 1909, xv, 75. Splenomegaly.

Oettinger, W., and Marie, P. L.: Rev. de Méd., 1911, xxxi, 345. De la Maladie de Banti.

Oller, D. A.: Rev. espec. med. Madrid, 1909, xii, 417. Tres casos de ruptura traumática del bazo observados en la clinica de Krönlein.

Orsos, F.: Virchow's Archiv. f. path. anat., 1909, cxcvii, 91. Beitr. z. Kenntnis der Wandermilz und der Splenomegale.

Ottario, C.: Gaz. d. osp. Milano, 1909, xxx, 594. Di un caso di accessoprimitivo della milza.

Otto, W.: Wiener klin. Wchnscht., 1910, xxiii. Three cases of splenectomy.

Papaioannon: Beitr. z. klin. Chir., 1910, lxx, 297. Ueber Splenektomie nach Milzruptur und Malariafieber.

Paulicek: Folio Hæmatolog., 1910, ix, Pt. 3.

Peterson, P.: Lancet, 1909, ii, 1496. Two rare surgical conditions.

Power, D'A.: St. Barth. Hosp. Rep., 1908, xliv, 95. Some cases illustrating surgery of the spleen.

Prince, E. M.: Alabama Med. Jour., 1910, xxiii, 210. Splenectomy in Splenic Anæmia, recovery.

Regling and Klunker: Deutsche Mil. Arzt. Zeitschr., July 5, 1911. Splenektomie wegen traumatische Milzruptur, Heiling, Blutbefunde.

Rendle, C. E. R.: British Med. Jour., 1909, i, 17. Traumatic rupture of the spleen.

Renton, J. C.: Lancet, 1909, ii, 82. Notes of a case of splenectomy for injury; with a report on the condition of the blood by J. M. Renton.

Rivet, A.: Gaz. méd. de Nantes, 1910, 2 s., xxviii, 21. Rate hypertrophique, flottante, avec torsion du pedicule, splenectomie, guérison.

Rivet, A.: Gaz. méd. de Nantes, 1910, 2 s., xxviii, 253, 417. Rate flottante; splenectomie guérison.

Rohrbach, W.: Wiener klin. Rundschau, 1910, xxiv, 247; 263; 278; 293. Ueber subkutane traumaticsche Milzruptur und deren Behandlung.

Royster, H. A.: Med. Record, Aug., 1911, 267. Three cases of non-parasitic cyst of the spleen.

Schulze, F.: Beitr. z. klin. Chir., 1911, lxxiv, 456.

Short, A. R.: Lancet. Sept. 16, 1911. Traumatic rupture of abdominal viscera.

Simonds, J. P.: Jour. Infect. Dis., 1908, v., xxiii. Splenomegaly and Banti's disease.

Siviatecki, J.: Medycyna i kron. lek., Warszaua, 1908, xliii, 1122. Three cases of splenectomy.

Smith, Mary A.: Annals of Surg., 1908, xlvii, 53. Splenectomy for carcinoma.

Solieri, S.: Archiv. f. klin. Chir., 1910, xcii, 479. Ueber operation an der Malariamilz.

Stirling and Grantley Sheldon: Austral. Med. Jour., n. s., i, 37. Splenic anæmia and splenectomy.

Strehl, H.: Archiv. f. klin. Chir., 1909, xxviii, 834. Ueber Milztuberculose.

Strina, F.: Ginecologia, 1910, vii, 526. Splenomegalia malarica e degenerazione cistica completa della milza in puerperio.

Summers, J. E.: Trans. Amer. Surg., Assoc., 1908, xxvi, 62. Œdema of the large intestine with localized necrosis of its walls, following splenectomy in Banti's disease.

Sutherland and Burghard: Lancet, 1910, ii, 1819. The treatment of splenic anæmia by splenectomy.

Swain, J.: Bristol Med.-Chir. Jour., 1910, xxviii, 223. A brief account of splenectomy with a case of simple hypertrophy of the spleen treated by operation.

Truelson, T.: Western Med. Rev., 1908, xiii, 514. Detailed report of a case of splenomegalia (Typus Gaucher) splenectomy; recurrence.

Viart: Bull. et mém. soc. de Chir. de Par., 1910, n. s., xxxvi, 594. Rupture traumatique de la rate; splenectomie, guérison (Rapport par E. Poterat).

Vickery, E. A.: U. S. Nav. Med. Bull., 1908, ii, 29. A case of Banti's disease with splenectomy and an arteriovenous anastomosis.

Vigard: Gaz. méd. de Nantes, 1908, 2 s., xxvi, 1055. Rate paludienne en ectopie pelvienne avec pedicule tordu; splenectomie; guérison.

Varwerk: Deutsche Zeitschr. f. Chir., 1911, cxi, 125. Subkutan. Zerreissungen der gesunden und kranken Milz.

Wallace, W. L.: Jour. Amer. Med. Assoc., 1907, xlix. Removal of a strangulated spleen in a child.

Wright, G.: Brit. Med. Jour., 1908, ii, 1809. Two cases of traumatic rupture of the spleen.

Zancan, A.: Policlinico, 1909, xvi, sez. med. 5. Un caso di morbo di Banti, splenectomia e guarigione.

CHAPTER IX.

TECHNIQUE OF OPERATIONS.

General Considerations.—In the first volume of this work we have covered so fully the subject of *preparation of the patient* and *after-treatment* that nothing additional need be said. In the **preparation of the abdomen,** however, we have somewhat modified our technique by adopting the *iodine method*, which for simplicity and efficiency seems to be unexcelled.

The abdomen is prepared preferably the evening before the day of operation. After the patient has been given a warm bath, assuming that his condition does not forbid the latter, the abdomen is shaved and washed with green soap and water, followed by a thorough rubbing with 65 per cent. alcohol. The abdomen is then covered with dry sterile gauze which is held in place by a light bandage. On the following morning the abdomen is painted with a 5 per cent. tincture of iodine, the field of operation is covered with a sterile towel and after the patient is etherized the abdomen is again painted with 5 per cent. tincture of iodine which is allowed to dry before the incision is made. In cases of emergency where immediate operation is imperative, the dry skin (without previous preparation) is painted with iodine tincture twice, each coat being allowed to become thoroughly dry. We have been able to observe no great difference in the effectiveness of the sterilization secured in this way compared with that secured by washing the abdomen several hours previous to operation with soap and water, but prefer to continue the latter method, as an additional safeguard, whenever time permits. Two things deserve to be emphasized: these are (1) that iodine applied to skin which has recently been wet is useless; and (2) that the iodine solution should be fresh, as the strength increases with age, owing to evaporation and concentration; in this way blistering of the skin may occur.

Operations on the Gall-bladder and Bile-ducts.

Position of the Patient.—The patient is placed in the dorsal position with a sand bag or firm pillow beneath the back at the level of the liver. This pillow should be about 18 inches long,

and cylindrical in shape; its diameter should be from 4 to 6 inches. Instead of a pillow a special operating table may be used, provided with an adjustable "bridge." When properly placed this pillow raises the lower dorsal and upper lumbar spine away from the table in such a manner that the liver and biliary passages are pushed forward, the costal angle is widened, and the intestines fall by gravity toward the pelvis. In this manner the bile-passages become more accessible. A pillow or two should be placed beneath the head and shoulders to relieve tension on the neck and to raise the head to a proper level for the administration of the anæsthetic. Elliot, of Boston, who introduced this position in 1895, also raised the head of the table (reversed Trendelenberg position); and in difficult cases this has much to commend it.

Incision.—Numerous incisions have been advocated as best adopted for exposing the gall-bladder and common duct. Some of these are objectionable, because disregarding important structures in the abdominal wall, especially the nerves, and thus predisposing to hernia or muscular atrophy. This danger from nerve injury was first pointed out by Assmy in 1899.

Courvoisier's Incision (1890) runs parallel to the costal margin from the ensiform to the flank; all structures of the abdominal wall are divided in the same line. Though this incision gives ample exposure it is objectionable because it cuts all the lower intercostal nerves.

Kocher's incision is much the same as Courvoisier's, but a little shorter and less transverse. It begins in the mid-line, below the ensiform, and crosses the rectus muscle obliquely to the semilunar line, being more distant from the costal margin here than at its commencement. All the structures of the abdominal wall are divided in the same line, but an attempt is made to preserve the nerves to the rectus muscle which cross the incision at the semilunar line. This is a good incision, but does not give sufficient exposure for severe cases.

Körte's Incision.—According to Sick, Körte formerly used the usual oblique incision, but now employs an incision which begins near the ensiform and goes obliquely downward and outward to the level of the umbilicus or lower. It is more nearly longitudinal than Kocher's, and after the sheath of the rectus is opened in this line, its fibres are split longitudinally.

Collins's incision is an improvement on Körte's. This incision "begins at the inner edge of the right rectus muscle 1 or 2 inches from the ensiform cartilage, and extends diagonally downward and outward to the outer edge of the right rectus close to the level of the umbilicus. It cuts through the skin, fat and anterior wall of the sheath of the rectus. A short transverse incision about 1 inch in length may be made inward from the upper end of the oblique incision through the skin, fat and linea alba; and a similar one through the linea semilunaris at the lower end. The rectus muscle is then separated from its sheath. When the muscle is thoroughly freed from its sheath, except at its outer border, it is easily retracted outward and allows the posterior sheath and the peritoneum to be incised in the same direction as the skin and anterior wall." Collins points out that transverse division of both linea alba and linea semilunaris permits the wound to gape widely, but that suture is easy and repair very efficient.

Lawson Tait and *Riedel* used a longitudinal incision through the right rectus muscle; and this is sufficient for ordinary cases of cholecystostomy.

Langenbuch's incision consists of a longitudinal cut along the right semilunar line, when necessary combined with an oblique incision along the costal margin to the ensiform; or a longitudinal incision through the rectus (as in Riedel's method), combined when necessary with an oblique incision along the costal margin inward to the ensiform or outward as far as necessary.

Mayo Robson's incision is similar to Langenbuch's second method –a longitudinal incision through the right rectus muscle, with an upward and inward extension along the costal margin to the ensiform. This is the incision we use habitually, and we find it adequate even in difficult cases. Yet Terrier, than whom no more skillful and experienced operator on the biliary tract has ever lived, declared this incision not sufficient, and for difficult cases preferred Kehr's incision.

Kehr's incision, known as the *Wellenschnitt*, and the *Bayonet incision*, begins at the ensiform, passes down 3 to 4 centimetres in the mid-line, then cuts the right rectus as far as its outer third (parallel to the costal margin) between its linea transversa and the umbilicus, and thence continues longitudinally downward as far

as the umbilicus or lower. There is no doubt that this immense incision gives very excellent exposure of the deeper biliary passages, but we have never felt inclined to adopt it, having found we could accomplish everything necessary through Robson's incision. If Kehr's incision is carefully sutured, and no wound infection occurs, it does not appear to predispose the patient to hernia more

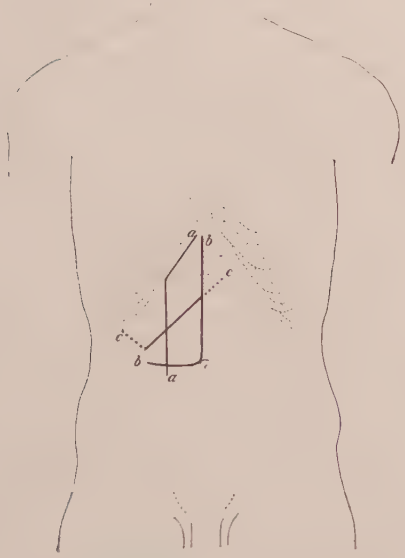

FIG. 32.—INCISIONS FOR OPERATIONS ON THE BILIARY TRACT.
aa. Mayo Robson's incision: *bb.* Czerny's Winkelschnitt: *cc.* Sprengel's Hakenschnitt.

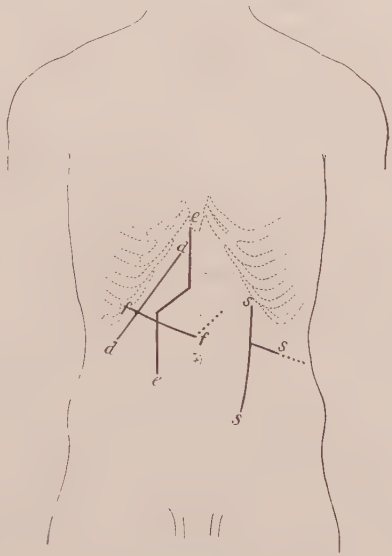

FIG. 33.—ABDOMINAL INCISIONS.
dd. Kocher's incision (Schragschnitt); *ee.* Kehr's Wellenschnitt: *ff.* Kausch's incision; *sss.* incision sometimes employed for operations on the spleen. An incision similar to Czerny's (Fig. 32) is preferable.

than the other incisions employed. It cuts no more nerves than Robson's incision, and divides the rectus in precisely the same manner, only a little lower. Of late Kehr has abandoned the lower limb of his original incision, so that the method he now adopts somewhat resembles that of Czerny.

Czerny's Incision.—Czerny described his *Winkelschnitt* in 1892; and Kocher has recently adopted it as his own "normal incision for difficult cases." The incision runs for from 5 to 7 centimeters in the mid-line, above the umbilicus, and bends just below the umbilicus to the right, cutting the right rectus muscle transversely.

Sprengel's Hakenschnitt consists of one limb parallel to the costal margin, and at the lower end of the first another shorter limb running upward to the costal margin at right angles with the first. The outer limb of the incision passes only through the external oblique aponeurosis, and does not divide the internal oblique or transversalis muscles. These, as well as the rectus and its anterior sheath, are divided parallel to the costal margin.

Sprengel's Transverse Incision.—Gosset, who with his chief, Terrier, had for years employed Kehr's Wellenschnitt, recently has adopted the *transverse incision*, a modification of Sprengel's incision described above. Gosset found that among 142 operations in which drainage had been employed after the use of Kehr's Wellenschnitt, a hernia resulted in four instances.

Sprengel's transverse incision divides the right rectus muscle directly across at whatever level seems desirable, depending upon the distance that the liver extends beyond the costal margin. If the suspensory ligament of the liver is in the way, it should be divided. Kehr objects to the use of this incision as consuming too much time. He speaks of fifteen or twenty minutes as required before the abdomen is opened. Gosset finds it takes him only from two to three minutes to make the whole incision. Only in exceptional cases is it necessary to divide part of the left rectus, to gain sufficient exposure. Körte has lately abandoned his own incision in favor of Sprengel's.

Kausch's incision begins at the costal margin in the mamillary line, and passes downward and inward to the mid-line just above the umbilicus. All the structures of the abdominal wall are divided in the same plane. He has employed this incision since 1899. It was adopted also by Mikulicz. In his early operations he employed an extension of this incision, in difficult cases, upward and downward in the mid-line; but he found extension downward was of no use, while sloughing of the point of the flap has followed extension directly upward. Now, in difficult cases he makes a similar oblique incision upward from the mid-line on the left; only once has it been carried as far as the left costal margin, usually only half across the left rectus muscle. This is the only incision which does not injure some nerves of the abdominal wall, except the incisions of Sprengel and that employed by Czerny;

and even the latter will injure some nerves if the transverse limb is very long.

Bevan's incision begins near the ensiform cartilage, runs parallel with the costal margin as far as the linea semilunaris, then extends along this line about 4 or 5 inches, and finally is carried transversely outward, thus making an elongated S-shaped incision. In simple cases neither the inner nor outer extension of the longitudinal incision need be employed. It is then the same as Langenbuch's original incision. When both the inner and outward extensions are used, it does more damage to the abdominal nerves than any incision, except Courvoisier's, ever devised.

As stated already, of all these incisions we prefer Mayo Robson's. In simple cases the longitudinal portion alone is used, splitting the fibres of the rectus muscle parallel to their course. If more room is needed, the incision is continued inward and upward, parallel but not too close to the costal margin, into the space between the ensiform process and the ribs. If the incision passes too close to the ribs difficulty will be experienced in suturing it, on account of the retraction of the flat muscles on the outer side of the wound. This incision of Robson's cuts only a few of the upper abdominal nerves, and only after they have entered the rectus muscle. Next to this incision we should prefer the Winkelschnitt of Czerny, which injures no nerves, and gives adequate exposure of the bile-ducts, pylorus and duodenum.

When the time comes to suture any of these upper abdominal incisions, closure is much facilitated by removing the sand pillow or support from beneath the patient, and thus relaxing the abdominal wall. When this is done, it should not be forgotten that the bile passages will recede at once from the anterior abdominal wall, and that the drainage may become displaced if not sufficiently long or firmly fixed.

After the abdomen has been opened, recognition of the anatomical landmarks and relative position of the viscera is of the greatest importance. This step in operations upon the biliary passages is often the most difficult one encountered throughout the entire procedure, especially in those cases where repeated attacks of inflammation have bound the various structures into an almost homogeneous mass by dense adhesions. These adhesions must be separated sufficiently to allow proper surgical treatment of

the lesions, but we do not advocate unnecessary interference with adhesions which often are nature's barrier against infection of the general peritoneal cavity. The adhesions should be approached from the convex surface of the liver, the border of the liver in the region of the normal site of the gall-bladder being freed first. From this point of entrance to the anatomy of the biliary passages, careful separation of the adhesions should be continued until the symptom-producing lesions have been uncovered.

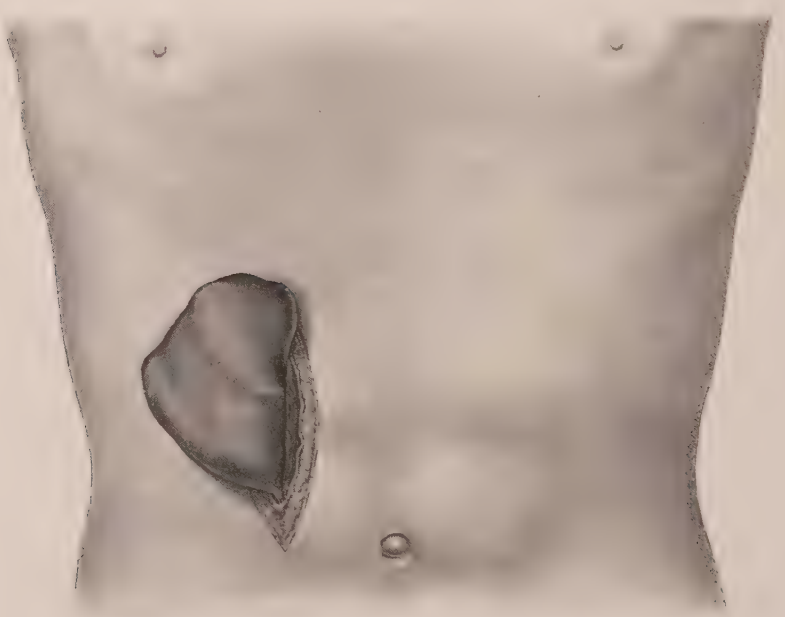

Fig. 34.—Rotation of the Liver to Expose the Gall-bladder and bile-ducts.

When possible, the lower border of the liver should be drawn slightly downward and then lifted upward into the abdominal incision slightly rotating the organ around an antero-posterior axis, so as to turn its inferior surface toward the left of the patient. This can be done in most cases where the anterior border of the liver is not held too firmly in place by adhesions. This lifting of the anterior border of the liver fully exposes the gall-bladder and the cystic and common ducts which are thus brought very near the surface. It may be noted as pointed out by Robson (loc. cit., page 250) that, "with the liver in this position, the cystic duct does

not form an angle with the common duct, but instead an almost straight passageway is found from the fundus of the gall-bladder to the entrance of the bile-duct into the duodenum." The ducts are fully exposed to view and with the palpating finger the surgeon may determine readily the condition of the ducts, the duodenum, the pylorus, the pancreas and the regional lymph-nodes.

Very few **special instruments** are required. For the removal of the bile from the gall-bladder a *trocar and canula* with a rubber tube attached may be employed. The canula should be large enough to permit the flow of thick, tarry bile. A *spoon scoop*, such as designed by Robson, is of great service in removing calculi from the gall-bladder or ducts. Large and small *gall-stone forceps* should also be provided. The ordinary small teaspoon answers admirably for removing stones from the gall-bladder.

Rubber tubing is needed in all cases of drainage of the biliary tract, either of the gall-bladder or the ducts. We prefer tubing with a lumen of about one-quarter of an inch for draining the gall-bladder; the tubing for the ducts should be small enough to enter the lumen of the duct easily, yet large enough to prevent leakage around the tube. A T-shaped piece of tubing is used at times to drain the common and the hepatic ducts as well as for reconstruction of the lumen of the common duct. The senior author has used for some years a specially constructed drainage tube for the gall-bladder. This is made by wrapping a thin layer of gauze around a rubber tube (1/4 inch lumen), and surrounding this gauze with rubber tissue or preferably rubber dam. The rubber tissue or rubber dam and gauze are held in place by catgut, tied around the whole in several places.

A sterile *hypodermic syringe* always should be part of the armamentarium in gall-stone operations, to be used to identify the common duct before incising it. This precaution of Terrier's will save the surgeon much uncertainty in cases where the choledochus is dilated and cannot otherwise be distinguished from the portal vein.

Cholecystendysis or Cholecystotomy without drainage has been abandoned by most surgeons, as the indications for its use are so seldom seen. The operation was first performed in 1883 by Meredith. Courvoisier performed it in 1884 under the name of cholecystendysis. Kocher is the principal advocate of its use today, claiming that the opening in the gall-bladder can be closed with

absolute safety if the proper sort of case is selected and if fine silk is used for the peritoneal stitches. For our part we think this practice is to be condemned. The supposed indication is to remove gall-stones from the gall-bladder, in the presence of absolutely clear and free cystic and common ducts, and in the absence of infection. It should not be performed if there is doubt about a calculus being overlooked either in the gall-bladder or in the ducts; nor if the lumen of the cystic duct, the common duct or the hepatic duct is even slightly narrowed by inflammatory deposits or cicatricial contractions; nor if the gall-bladder walls or its contents show any evidence, however slight, of infection.

In *exploratory operations* upon the gall-bladder, it may be desired to know the general characteristics of the bile, and without making a formal opening into the organ, a hypodermic needle may be inserted and some of the bile aspirated for study. The opening made by the needle requires no suture.

Operation.—The gall-bladder is exposed and isolated by gauze packs, as in other operations upon the biliary tract. Its fluid contents are removed by aspiration, it is opened, and the calculi removed. The wound in the gall-bladder is then closed with a row of plain catgut sutures which pass through all of the coats of the viscus, and these are reinforced by a continuous Lembert suture, of chromic or iodized catgut. Silk should not be used for fear of its ulcerating into the lumen of the gall-bladder and forming a nucleus for a stone. The gall-bladder is dropped back into place, the gauze pads are removed, and the wound in the abdominal wall is closed in tiers without drainage. It is always advisable to use two or more interrupted silkworm-gut sutures running down to the peritoneum through all layers of the abdominal wall ("splint sutures") as advised in Vol. 1 (page 347).

Cholecystotomy and Cholecystostomy.—The two terms, cholecystotomy and cholecystostomy, are used as synonyms by most authors and writers. Mayo Robson (loc. cit., page 270) makes no distinction. On historical grounds it might be preferable to employ the term cholecystotomy to designate solely the operation of opening and draining the gall-bladder as usually done, and to reserve the term cholecystostomy to designate the operation of draining the gall-bladder by attaching it to the parietal peritoneum or anterior sheath of the rectus, as is done when very prolonged

drainage is desired (cases of pancreatitis, etc.); but the terms are in such general use as synonyms that the distinction is of questionable value. In many cases the drainage of the gall-bladder is merely incidental to a more important procedure, such as removal of calculi, or evacuation of an empyema; in such cases opening and not drainage of the gall-bladder constitutes the main therapeutic indication. Cholecystendysis is best reserved to describe the suture and replacement of the gall-bladder without drainage. Mayo Robson, even as late as 1909, still recommends that the gall-bladder should be stitched to the aponeurosis of the rectus whenever possible; only when the gall-bladder is atrophied and shrunken, and cannot be brought up into the abdominal incision is he satisfied to drain it as here advised.

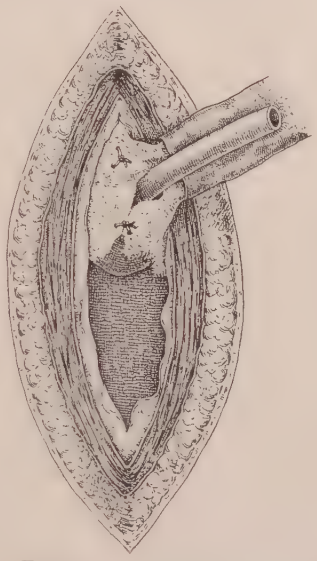

Fig. 35.—Cholecystostomy: Drainage Tube in Gall-bladder and Gauze Ends of Cigarette Drain Sutured to Fundus of Gall-bladder.

Operation.—In most cases a simple longitudinal incision, 3 or 4 inches long, through the outer third of the rectus muscle is sufficient. It is wise, particularly in jaundiced cases, to clamp and ligate all superficial vessels as they are cut, to prevent undue bleeding.

The opening in the peritoneum is made large enough to permit proper exposure and manipulation of the viscera. After the wound has been separated with retractors, the field of operation is inspected and explored with the hand. Then a large gauze pad wrung out of hot salt solution is packed down into the subhepatic space or right kidney pouch. A second pad of similar character is packed toward the mid-line walling off the stomach. A third pad should be placed between the gastro-hepatic omentum and the left lobe of the liver. A fourth pad is then packed down toward the cæcum. A fifth pad or a gauze sponge may be passed up over the right lobe of the liver to protect the subphrenic space.

The field of operation is thus walled off from the rest of the

peritoneal cavity, and any leakage of bile or infective material will not extend to other parts.

Adhesions should then be looked for, and separated if present. Some will yield readily to the finger, while others must be clamped, cut and ligated. Omental adhesions always should be clamped, cut and ligated, for fear of subsequent hemorrhage.

Now the gall-bladder should be brought into the wound, and if this is not readily done, the right lobe of the liver may be pulled down and rotated out of the wound, as already described, to give a better exposure (Fig. 34). The gall-bladder is then seized at the fundus, and pulled into the wound with the fingers, using a piece of gauze to keep the fundus from slipping away; if this cannot be done with the fingers, forceps may be used. The surrounding skin and wound margins are then protected with gauze.

If the condition is one of hydrops or empyema of the gall-bladder, with marked distention of the organ, it should be emptied by aspiration by means of a trocar and canula. To the end of the canula a rubber tube is connected, permitting the contents of the gall-bladder to flow into a receptacle and preventing them from contaminating the wound. The fluid should be sent to the pathological laboratory for culture. The gall-bladder is then held up by means of forceps and is opened at the fundus with scissors, permitting the insertion of the finger. The interior of the organ is then explored with the finger, the general appearance and condition of the mucosa and the presence or absence of stones are noted. Stones and inspissated bile are removed by means of scoop, spoon or forceps. Before concluding the operation, unless the patient's condition is critical, the surgeon should examine the common duct and the pancreas. During this examination the gall-bladder is temporarily plugged with gauze. To expose the common duct, the gall-bladder is pulled upon putting the cystic and common ducts on the stretch. The cystic and common ducts may then be examined by sight and touch, the index-finger of the right hand being passed into the foramen of Winslow, while the thumb is placed on the free border of the gastro-hepatic ligament. Kehr advises the surgeon to use the left hand, standing with his back toward the patient's head, and keeping his hand fully pronated during this digital exploration of the ducts; but usually we prefer not to change our position, since sight can be

used as well as touch. If a stone is present in the common duct, it may be possible to push it along the choledochus into the duodenum; or it may be removed by a scoop or gall-stone forceps passed down into the duct through the gall-bladder and cysticus, if the latter is much dilated. When these methods are ineffective, as they often are, the common duct must be opened in a manner that will be described later.

No gall-bladder operation is complete without examination of the common duct and of the pancreas, and searching for enlarged peripancreatic lymph-nodes. At this stage of the operation, the pancreas should be palpated throughout its entire length, the size and consistency of the organ being noted. Enlarged lymph-nodes should be looked for, particularly along the upper border of the pancreas (gastro-hepatic omentum), at the junction of the cystic and common ducts and at the junction of the supra- and retroduodenal portions of the common duct.

The gauze is then removed from within the gall-bladder and its interior again explored with the finger. A purse-string seromuscular suture of plain catgut, No. 0, is then passed around the gall-bladder about an inch from the opening and left untied. The special gall-bladder tube (page 447) is then inserted into the opening for a distance of about an inch. The edges of the gall-bladder opening are then fastened to the sheath of the tube with two or more catgut sutures. These sutures control the bleeding that would otherwise occur.

The gall-bladder is then grasped with tissue forceps at two opposite points, below the purse-string suture and the tube is invaginated taking with it the edges of the gall-bladder. The purse-string suture is then tied. To insure a good invagination a little instrument such as that used by Mayo to tuck in the edges evenly often is of use. The advantage of this procedure is that a valve-like closure of the opening in the gall-bladder is formed, so that after the tube is withdrawn the fistula closes rapidly.

It may prove impossible to invert a portion of the wall of the gall-bladder in the manner recommended, if the gall-bladder is small or has contracted and thickened walls. Then the opening in the gall-bladder should be sutured as tightly as possible around the tube. In such cases, or whenever there is uncertainty as to the security of the attachment of the tube to the gall-bladder, it

is safer to insert also a small cigarette or rubber drain beside the gall-bladder, to provide an exit for leakage. This may be attached by one or two sutures to the fundus of the gall-bladder (Fig. 35). The gall-bladder tube is made of such a length that 2 or 3 inches project from the abdominal wound or through a stab wound made to the outer side of the former—thus allowing the abdominal wound to be closed throughout. This tube remains until it comes away of its own accord, which is usually about the fourteenth or sixteenth day. At times a plain rubber tube is used in place of the special gall-bladder tube; this is inserted in the same way.

All gauze pads and sponges are now removed from the abdomen, the gauze count made and the instruments accounted for. The sand pillow is removed from beneath the patient, as the closure of the wound is facilitated by its removal. The wound is then sutured from its upper and lower angles, toward the point of exit of the tube if the latter is brought out through the wound. The tube is always given exit from the wound where it will afford best drainage; at times, as indicated above, a counterpuncture is necessary to insure good drainage. This depends largely upon the size and position of the gall-bladder. When a separate incision is required for the drainage it usually is made to the outer side of the operation wound, which may then be closed completely.

The abdominal wound is closed as recommended in Vol. I, page 347. A glass elbow is then fastened in the projecting gall-bladder tube and is connected with a bottle to collect the bile. This bottle may be carried in the dressings, beneath a Scultetus bandage, or the bottle may be hung by the side of the bed, a long tube being carried to it from the wound. A dry gauze dressing is then applied with a proper retentive bandage.

Cholecystostomy with prolonged Drainage.—It is especially in cases of cholangeitis, pancreatic lymphangeitis, and chronic interstitial pancreatitis, that this method is advisable. It may require many months for the biliary tract to become sterile.

The *operation* differs in no material respect from that of the ordinary cholecystostomy already described, save in the fact that the gall-bladder is sutured either to the parietal peritoneum or the anterior sheath of the rectus, and is not dropped back into the abdominal cavity after the margins of the opening in its fundus have

been inverted around the drainage tube. The other manipulations having been completed, the gall-bladder tube is inserted for about an inch into the opening in the fundus of the gall-bladder and is fixed to the margins of this opening with one or two sutures of plain catgut (No. 0). The opening in the gall-bladder is then closed tightly around the tube, with sutures; but if a purse-string suture is used, no effort should be made to invert the edges of the gall-bladder opening, as the purpose of the operation is to secure prolonged drainage and prompt closure of the fistula is undesirable.

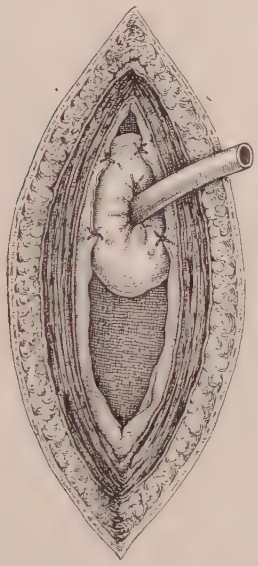

FIG. 36.—FUNDUS OF GALL-BLADDER SUTURED TO THE PARIETAL PERITONEUM, TO SECURE PROLONGED DRAINAGE.

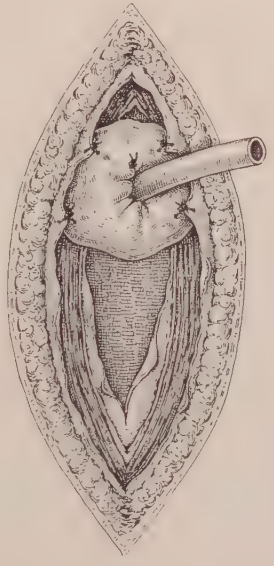

FIG. 37.—FUNDUS OF GALL-BLADDER SUTURED TO THE ANTERIOR SHEATH OF THE RECTUS MUSCLE, TO SECURE VERY PROLONGED DRAINAGE.

When the abdominal wound is being closed, the fundus of the gall-bladder is caught by at least two sutures passing through the parietal peritoneum on both sides of the abdominal incision, so as to anchor the gall-bladder firmly in the wound (Fig. 36). If very prolonged drainage is desired the gall-bladder is sutured to the anterior sheath of the rectus muscle (Fig. 37). As already noted, Mayo Robson, as late as 1909, still advocates this method of treatment of the gall-bladder, even in cases of simple cholelithiasis, where prolonged drainage is not required; and he recommends

only in exceptional cases, where this method proves impossible, the procedure we have described as simple cholecystostomy—dropping the gall-bladder back into the abdomen after closing its opening around a drainage tube.

Cholecystectomy.—In this operation the patient is placed in the usual position, the same incision is made, and the parts are walled off with gauze as described. The best method of removing the gall-bladder is to work from the cystic duct and artery toward the fundus. This is preferable to working in the opposite direction in that the vessels are clamped and ligated first, which is no doubt the most important and at times the most difficult part of the operation. Moreover, when working from the fundus downward to the duct and artery, blood from the denuded surface of the liver may run down and obscure the view, and time is lost in sponging it away.

The liver is pulled down and rotated out of the wound, when this is possible, and is held by an assistant. The cystic duct is located and isolated by making a small incision through the peritoneal covering overlying its termination. The cuff of peritoneum is wiped with wet gauze toward the common duct, exposing the cystic duct and vessels. The cystic duct, close to its termination in the choledochus, is grasped between two hæmostatic forceps, of which the curved type proves most satisfactory for this purpose. The duct is then divided between the hæmostats. Care must be taken not to include a part of the wall of the common duct. The cystic artery and vein which lie above and to the inner side of the duct are then clamped with two hæmostats and are divided between them. At times it will also be necessary to clamp and ligate an anomalous branch of the gastro-duodenal artery which supplies the cystic and common ducts. The stumps of the duct and vessels may be ligated at this time, or if so desired not until the gall-bladder is removed. In either case iodized catgut, No. 2, is used. It is a good plan, before ligating the stump of the cystic duct, to explore the common and hepatic ducts as described below. When there is doubt as to the need for drainage through the stump of the cysticus, this may be clamped by a hæmostat, as suggested by Ochsner; the hæmostat is surrounded by gauze and rubber protective, and left protruding from the wound. This clamp may be removed in thirty-six hours, or even sooner if it is decided

that drainage is required. Formal drainage is described at page 461. The method of draining the hepaticus after cholecystectomy is described at page 457.

After the stump of the cystic duct and the cystic vessels have been ligated, the separation of the gall-bladder from its fibrous bed is begun. This is done in such a manner as to preserve, if possible,

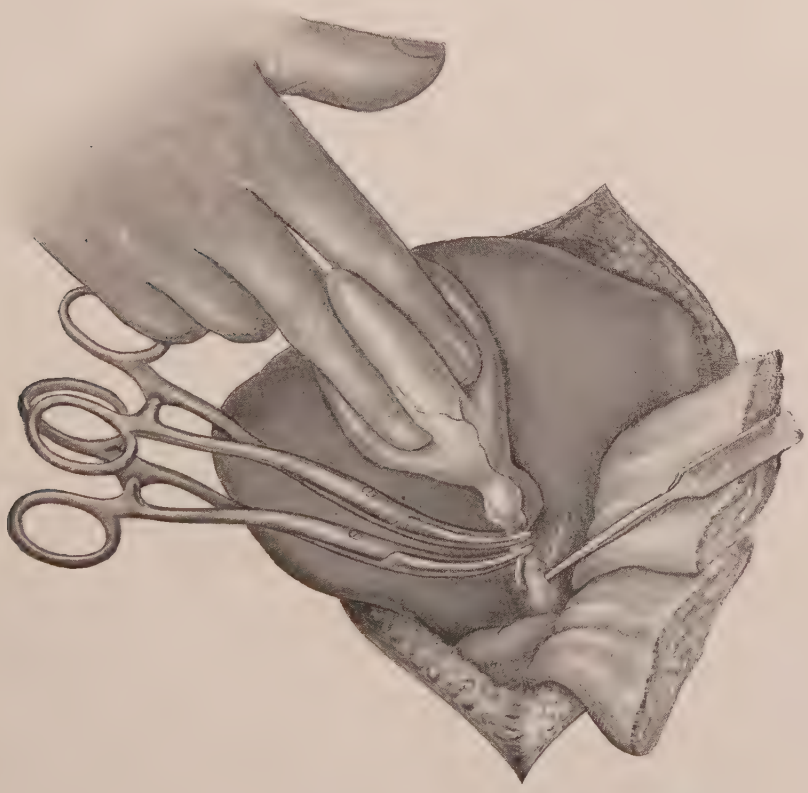

Fig. 38.—Cholecystectomy: Cystic Duct Isolated and Clamped.

that portion of the fibrous bed immediately adjacent to the liver. By means of the finger the gall-bladder is stripped toward the fundus, cutting with scissors the peritoneal fold when necessary. When the separation is done carefully, there is very little if any bleeding from the liver substance. The peritoneal fold is then closed by a continuous suture of iodized catgut, unless the hepatic surface is infected, when the gall-bladder bed should be drained.

The peritoneal cuff overlying the stump of the cystic duct is closed with a single chromic catgut suture.

Terrier's Operation.—In cases where *very dense adhesions* exist, it may be very difficult to identify the cystic duct before isolating the gall-bladder. Under such circumstances we believe the best technique is that systematized by Terrier: The anterior margin of the liver is identified, and the fundus of the gall-bladder found.

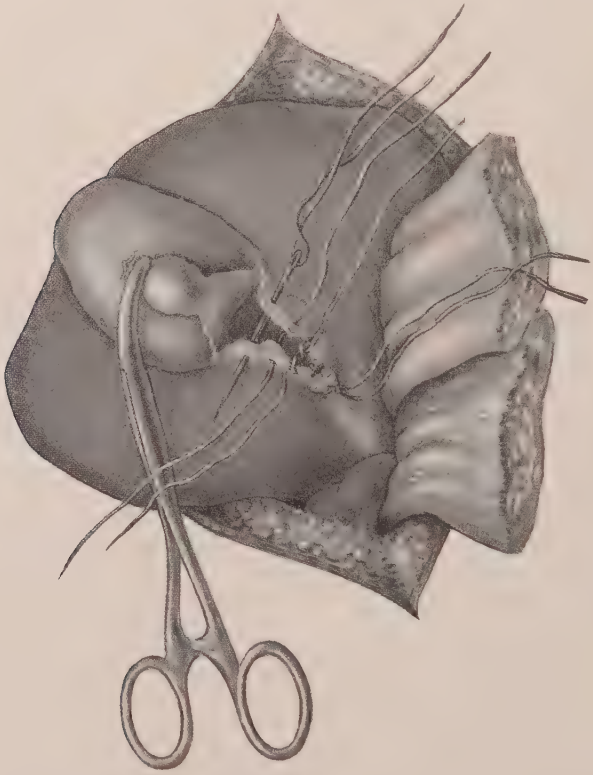

Fig. 39.—Cholecystectomy: Dissection of the Gall-bladder from its Hepatic Attachments.

The gall-bladder is then opened at its fundus and its inferior wall is cut open little by little by snipping with fine scissors; forceps are clamped on bleeding points in the gall-bladder wall. This incision is continued into and through the cysticus, right down to the choledochus. This splitting of the cystic duct is the only

difficult part of the operation, and it may be very difficult. The duct cannot be distinguished from the outside, on account of adhesions; and it is only recognizable because the operator cautiously follows its lumen, much as one follows the lumen of a strictured urethra in performing external urethrotomy without a guide. When the strictured cystic duct has been split all the way down to the choledochus, the cystic artery is ligated, and the gall-bladder and cystic duct are removed.

The common duct and the hepatic duct are then explored (by different sounds, so as not to carry duodenal infection into the hepaticus), and the hepaticus is drained through the opening left by excision of the cysticus. A rubber drainage tube, or a soft rubber catheter (No. 25 to 35 Fr.), open at the end, but having no lateral fenestrations, is passed up the hepaticus to its bifurcation, where it is felt to be arrested. The tube is then withdrawn slightly, and is stitched in this position, with No. 0 chromic catgut, to the stump of the cysticus, in such a way as not to diminish the lumen either of the duct or the tube. The tube is brought out of the abdominal wound at the most convenient point; and should be carefully distinguished (by color, by the insertion of two safety pins instead of one, or in some other way) from any tube used to drain the subhepatic space.

In cases where the removal of the gall-bladder is attended with much bleeding from the liver substance, this usually may be controlled by suturing tightly together the peritoneal folds which covered the gall-bladder, thus compressing the raw surfaces of the gall-bladder fossa. These sutures, of heavy iodized catgut, should be passed with a large curved needle, and should pass into the liver substance. At times a split rubber tube with a piece of gauze, or a cigarette drain (a rolled piece of gauze surrounded by rubber tissue), may be placed in the fossa, and sutured in place as above described, thus exerting sufficient pressure on the bleeding surface to check all hemorrhage. These drains if used are allowed to remain until they come away easily, with not much pulling, which usually is about the sixth or the seventh day. It is very important in this operation to control all bleeding before closing the wound, and especially is this so in cases with jaundice. Of late we have abandoned the use of the split rubber tube carrying a gauze strip as we find it possible to control the bleeding with sutures carried

into and around the gall-bladder bed. In highly infectious cases, however, it is safer to drain the gall-bladder bed.

After the gauze is removed from the abdomen and the viscera placed in their proper position, it is wise to place a rubber tube in the subhepatic space or right kidney well, as far as the posterior abdominal wall. This tube should have a diameter of about one-quarter of an inch and should be fenestrated near its lower end. The function of this tube is to drain off any blood or bile which may have gravitated to this space during the operation, or which may subsequently collect there. The tube is left projecting from the abdominal wound, or stab wound, for about an inch, and through the projecting end a safety pin is passed to prevent the tube from falling into the abdominal cavity. This tube is removed thirty-six hours after operation, and it is not necessary to replace it with any form of drainage.

In cases where there has been considerable hemorrhage or escape of bile into the subhepatic space, it is well to use a glass drainage tube instead of the rubber tube just advised. This tube should be aspirated and turned every three hours or oftener if necessary, and should be allowed to remain for thirty-six or forty-eight hours, or until the drainage from it becomes clear and straw colored. When this tube is removed, it is not replaced with any other form of drainage, except when the material drained is shown by culture or is believed to be infective; in which case a rubber drainage-tube should replace it, and this should be retained until purulent discharge ceases. When a rubber is used to replace the glass tube, it should be passed down through the glass tube and the latter removed by withdrawing it over the rubber tube.

The wound after cholecystectomy is closed around the drainage in the manner which has been described in the previous operations; the tube which drains the hepaticus should not be removed for at least two weeks (page 462); it is best to allow it to remain as long as it will.

Choledochotomy.—As we have already stated (page 128) most surgeons employ the terms *choledochotomy* and *choledochostomy* interchangeably. Strictly speaking, the former implies merely an incision into the common duct for the purpose of removing a calculus; while choledochostomy, in its historical sense, indicates suture of the dilated choledochus to the abdominal wound for the

purpose of more or less permanent drainage. As the former operation (choledochotomy), however, almost always is supplemented by drainage of the duct by tube, and as closure of the choledochus by suture, without drainage, is now an operation only of historical interest, any distinction between the terms choledochotomy and choledochostomy seems a refinement.

These may be comparatively easy operations or the most difficult operations in surgery. The difficulties are due to strong adhesions binding the surrounding structures together into a mass which destroys all anatomical landmarks. Especially difficult is the discovery of the choledochus if the gall-bladder and cystic duct have been removed at a previous operation, since there is then no sure guide to follow. The skill of the surgeon will be taxed to the utmost in many cases where the abdominal walls are very thick and the liver is so fixed by adhesions that it cannot be dislocated and brought into the wound. In thin patients, with relaxed abdominal walls, especially women with movable liver, and where no adhesions are present, rotation of the liver is very easy, and the common duct can be brought up into the abdominal incision, permitting all subsequent manipulations to be carried on in full view. In other cases, where the liver is fixed, the difficulties appear to be unsurmountable, but they can be overcome without irremediable damage to the parts, if only proper carefulness and patience are exercised, and the operator possesses sufficient surgical skill. Excellent illumination, and especially the Elliot position, do much to lighten the burden of operating.

Choledochotomy when the Gall-bladder is Present.—The usual incision is made through the abdominal wall, all adhesions are separated, the liver is drawn downward and upward into the wound and rotated. After careful examination of the gall-bladder and ducts has been made for the purpose of locating stones and determining their presence in the common duct, the gall-bladder is tapped and all fluid withdrawn, all precautions against soiling the abdomen, as described at page 449, being carefully carried out. The gall-bladder is then opened and freed from calculi and débris. We believe that this step in the operation is advisable in all cases where the gall-bladder is distended, with a patulous cystic duct, as it will relieve some of the tension in the common duct and prevent excessive escape of bile from

the choledochus when this is incised. The position of the calculi in the common duct is then determined by the palpating finger which may reach almost all portions of the duct when passed through the foramen of Winslow. When possible, the calculi should be carefully worked backward into the supraduodenal portion of the duct, if they are found below this portion; or they should be pushed downward into it from the hepaticus, as the common duct can be opened with most facility and safety in its supraduodenal course. Trouble may be experienced in some cases in determining the exact location of the duct and distinguishing it from the portal vein. We long ago adopted the following method of distinguishing between the two; we are confident it is safe and sure, and we are pleased to know that so skillful an operator as Terrier employed it as a matter of routine in difficult cases. The free border of the gastro-hepatic omentum is carefully incised, a small hypodermic needle is thrust into the lumen of the structure supposed to be the common duct, and the barrel of the syringe is filled with the contents of the structure. The appearance of the fluid withdrawn will show at once whether it is bile from the duct or blood from the vein. The minute puncture immediately closes after withdrawal of the needle; we have never noticed any leakage from such a puncture. It is not an uncommon anomaly for the cystic duct to be inserted at a very oblique angle, and very low in the common duct; thus for some distance the cystic duct runs parallel to the hepatic duct, within the gastro-hepatic omentum. When a stone is present in the supraduodenal portion of the duct, it is grasped between the finger and thumb and an incision is made through the walls of the duct, in the direction of its long axis, directly over the stone, and of sufficient length to permit easy removal of the calculus. The duct is carefully explored with the finger when the duct will admit its entrance, or with a probe or scoop for the purpose of determining the presecne or absence of other stones. When they are present, they should be pushed toward the opening in the duct. When this is not possible they must be removed by one of the methods described below. When the stone is friable, it may be crushed between the finger and thumb, the fragments being extracted with the scoop through the original incision in the duct. This procedure may cause too great trauma

to the walls of the duct, but we believe that crushing with the finger and thumb, or with the gall-stone scoop, if the proper care is exercised, will do less damage and expose the patient to less risk than the other methods to be described. When the above procedure will not suffice, it may be possible, by the aid of the gall-stone scoop, to push a calculus or several calculi onward into the duodenum.

Thorough exploration of the hepatic duct must be made through the incision in the common duct, the probe being passed into each branch. This probe should be perfectly clean, not one that has been passed before into the choledochus. The probe or small scoop should then be passed into the duodenum for the purpose of determining the patency of that portion of the duct. Occasionally a caluclus which cannot be detected in this way may be felt by a finger inserted into the dilated duct. Numerous cases have been reported where calculi have been left behind, repeated operations being required to relieve the patient of stones which should have been removed at the first operation. Concretions have been left behind by almost every surgeon who has had a large experience in gall-stone surgery, and such cases have been reported by Robson, Riedel, Kehr, Terrier, Fenger, Küster, Lauenstein and numerous others; and both of the present writers have committed similar offenses. Unless all concretions are removed, the operation will not afford permanent relief.

After the duct has been cleared it should be drained. A small rubber tube is introduced into the duct and gently passed toward the hepatic duct. When the duct is friable or does not seem to be very healthy, or where sutures may not readily be introduced for the purpose of closing the opening in the duct, a T-shaped tube is used, one arm extending toward the duodenal opening of the duct. The latter arm must not be so long as to extend into the duodenum, otherwise some of the duodenal contents will escape by way of the tube, creating a duodenal fistula. We believe that the opening in the choledochus never should be closed, but should be drained in all cases. After the drainage tube has been properly placed, it is held in position with a small chromic catgut suture and the opening in the duct is closed with catgut sutures around the drainage tube, thus preventing leakage of the infectious bile into the abdominal cavity. The tube in the choledochus should be left in place until it comes

away of itself. This usually is during the third week after operation. The T-tube can be left for three weeks or longer, if thought best. There is no difficulty in removing these tubes after that lapse of time—they come away quite easily.

In almost every case of choledochotomy, the gall-bladder, when present, will have been opened for purposes of exploration and for the removal of calculi, as indicated already. At the conclusion of the operation upon the common duct, the gall-bladder should be drained as described at page 451 (Fig. 35). If cholecystectomy is indicated the stump of the cystic duct may be closed by sutures.

Drainage of the subhepatic space (page 458) by rubber tube or cigarette drain is indicated in every operation upon the common duct.

Choledochotomy when the Gall-bladder is Absent.—This usually is a difficult operation. A cautious and lengthy dissection may be necessary, to separate adherent stomach, duodenum, omentum, or colon, from the under surface of the liver, and to enable the surgeon, finally having passed these adhesions, to expose the common duct. It is tedious and difficult, but usually possible to expose the supraduodenal portion of the choledochus in this way. When the common bile-duct has been found, it is opened in the usual way, and the operation concluded as already described. Desjardins recommends that the operator commence by exposing the choledochus in the retroduodenal portion, by mobilization of the duodenum. He thinks that in this way time is saved, and there is less uncertainty about the position of important structures. There are cases in which it may seem desirable to incise the duodenum and identify the choledochus by retrograde catheterization through the ampulla of Vater. This method has been employed once by the senior author:

Obliteration of Choledochus Following Cholecystectomy; Retrograde Catheterism and Drainage. Death.

Ella L., aged thirty-six years, admitted to the German Hospital, December 1, 1911. Complaint, jaundice and pain in upper right quadrant of abdomen.

Previous Medical History.—Patient always nervous and never strong. Inflammatory rheumatism at thirteen. Scarlatina at six. Pleurisy at sixteen. Tonsillitis regularly once a year up to fourteen years. Seldom has colds.

Present Illness.—Ten to twelve years ago began to be troubled with pressing sensation in pit of stomach, distention, belching and pain in upper

right quadrant of abdomen, coming on about three hours after eating. Sometimes free from pain for four months at a time.

One year ago began to have attacks of severe pain in gall-bladder region, radiating to right shoulder, and sometimes from left costal margin to cardiac region; then sharp pain would radiate over whole body. Had four such attacks. Operation on February 18, 1911— cholecystectomy for gall-stones, also appendectomy. No jaundice or sweats before operation; drained for fourteen weeks following operation. Then well for four weeks. Two weeks after returning home had a second attack of jaundice and nausea. Urine dark, and since drainage has stopped skin has had light yellow tinge. Bowel movements eight or nine times daily. Itching all summer. Two weeks ago had a cold and cough with pain in right hypochondrium and costo-vertebral angle, not sharp and not nauseating. No urinary symptoms. No chronic cough. Some pain in right leg. Condition same during last week.

Physical Examination.—Poorly nourished female. Marked jaundice of scleræ and general jaundice.

Heart: Muscle sound not very strong.

Abdomen: Distended, slight right-sided rigidity. Numbness on pressure over mid-epigastrium. Bimanual palpation reveals large liver, but whether all of tumor mass in upper right abdomen is liver cannot be determined.

Operation.—December 2, 1911. Common duct not found; probe passed through diverticulum of cystic duct passed only a short distance and became obstructed; unable to find remains of common duct; probe passed upward into hepatic ducts without difficulty, pylorus isolated; intestines walled off well, the duodenum then opened; ampulla of Vater found and probed passed, but only a short distance in the lower portion of the gastro-hepatic omentum —probably the distal portion of remains of common duct. A large probe now passed through distal end of common duct into duodenum; still larger probe used in similar manner, but it became obstructed at ampulla. The ampulla was then incised and large probe passed freely. T tube inserted, one end in distal extremity of common duct and other end in diverticulum of cystic duct; sutured in place with chromic gut. One end of tube passed up through diverticulum into hepatic duct. Duodenum now closed with iodine gut followed by two layers of continuous linen. One spiral tube drain down to site of operation. Wound closed with chromic gut; two through and through silk-worm gut sutures; skin closed with silk-worm gut. Iodine dressing.

Following operation patient complained of weakness although general condition remained good.

Pulse of fair volume. Jaundice much improved.

December 7, 1911.—Slight bleeding from wound which was readily checked by Monsel's solution.

December 8, 1911.—At 9.30 P. M., patient vomited 500 c.c. of dark grumous liquid—looks like blood. Pulse rapid and thready. Abdomen soft, no distention. Fair peristalsis. No evidence of peritonitis or obstruction. Transfused and active stimulation given—patient greatly improved. During

the night patient vomited bright blood and expelled blood per rectum. Origin of bleeding looked for but not discovered.

December 9, 1911.—Died 10.20 A. M. Wound opened and stomach found distended with yellow fluid. Site of operation in excellent condition. No evidence of peritonitis. Mesentery of the ileum contained an ecchymosis a little larger than a dollar. Near the mesenteric attachment a distended blood-vessel, apparently thrombotic, stands out prominently. The small intestines contained blood.

Patient evidently died as a result of the hemorrhage which in her greatly weakened condition she was unable to withstand.

Transduodenal Choledochotomy.—This term is applied to the operation of choledochotomy when the calculus is removed from the choledochus through an incision made in the duodenum. The operation was first performed by McBurney, in 1891, followed by Kocher, in 1895, and by Mayo Robson in 1898. It is applicable to cases of impaction of a calculus in the retroduodenal, pancreatic, or interstitial portions of the common duct. The interstitial portion includes the ampulla of Vater, a dilated portion of the duct in which a stone may become lodged and from which it can only with great difficulty be dislodged either into the duodenum or back into the pancreatic portion of the duct.

The abdomen is opened and the biliary passages liberated from adhesions and if possible brought into the wound in the usual manner. The duodenum and the terminal portion of the duct are raised by the fingers of the left hand, and the anterior wall of the duodenum is incised. McBurney advised a transverse incision, and this was employed by Kehr. We believe that an incision parallel to the long axis of the bowel is preferable, as it gives better exposure of the structures within the lumen of the intestine and is more easily closed with less danger of leakage or subsequent stricture. After the duodenum has been opened, the papilla is to be located; if a stone is found within the ampulla the biliary orifice is either dilated or incised upward, and the stone removed. When the stone is lodged in the pancreatic or retroduodenal portions of the duct, the posterior wall of the duodenum may be incised, thus exposing the wall of the choledochus; the stone is then removed through an incision made into the duct directly over the stone. When the papilla has been dilated or incised, no attempt is made to close the opening. When the posterior wall of the duodenum and the common duct have been incised separately it is advisable to

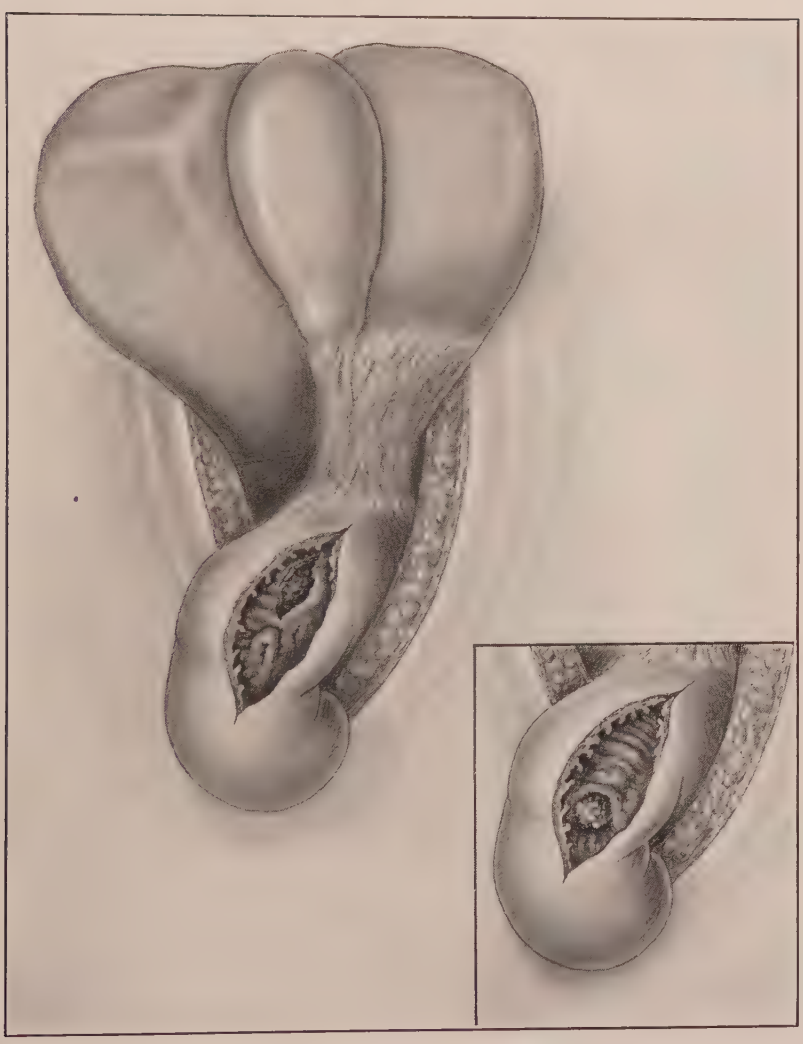

Fig. 40.—Transduodenal Choledochotomy.

In the Larger Drawing the Retroduodenal Portion of the Choledochus has been Incised over an Impacted Stone by means of an Incision in the Posterior Wall of the Duodenum, Exposed by Opening its Anterior Wall (Kocher's Operation).

The smaller drawing shows a calculus protruding into the duodenum from the ampulla of Vater.

suture the opening in the duct to the incision in the posterior wall of the duodenum in such a manner as to form a fistula. This operation was introduced by Kocher (1895) and is named by him *duodeno-choledochostomy*, thus distinguishing it from *duodeno-choledochotomy*, the operation of McBurney.

The incision in the anterior wall of the duodenum is closed with catgut, with a reinforcing suture of linen. If the choledochus has already been opened in its supraduodenal portion for the purpose of removing a stone, it should be drained in this situation; but if it has been opened only by the transduodenal route no external drainage of the duct is necessary. The operation is then completed in the usual manner.

The senior author, in his entire experience, has been forced only once to adopt the transduodenal method of removing a calculus from the common duct. In all his other operations it has been possible either to extract the stone (entire or after breaking it with the gall-stone scoop) through an incision in the supraduodenal choledochus or to push the calculus into the duodenum by manipulation with the finger or the gall-stone scoop.

Retroduodenal Choledochotomy.—When a stone is impacted in the retroduodenal or pancreatic portions of the common duct, and cannot be dislodged, this part of the duct may be exposed by **mobilization of the duodenum**. The operation should not be regarded as a substitute for duodeno-choledochotomy which should be reserved for stones impacted in the ampulla of Vater or very close to the duodenal wall. Mobilization of the duodenum is a procedure which consists in separating the duodenal loop from its secondarily acquired attachment to the posterior parietal peritoneum, and restoring it to the state in which it existed during fœtal life, with a distinct mesentery of its own (Vol. I, Fig. 3). The right side of the primitive duodenal mesentery becomes adherent to the posterior parietal peritoneum overlying the right kidney; and in extrauterine life this adhesion becomes so intimate that no visible indication exists of the former state of affairs. If, however, an incision is made just to the right of the descending portion of the duodenum, through the parietal peritoneum overlying the right kidney, it becomes possible by blunt dissection, and with very trifling hemorrhage, to separate the duodenum with its primitive mesentery (which carried the gastro-duodenal vessels) from the original

posterior parietal peritoneum, which covers the kidney and vena cava, and thus to restore to the duodenum its former mobility. This is the same principle which is used in mobilizing the sigmoid in resection of the colon, for which operation the method was introduced by Pierre Duval. The idea of thus mobilizing the duodenum originated with Terrier, according to Leriche, and was described by his pupil Jourdan in 1895, and by Vautrin in 1896; it is the same

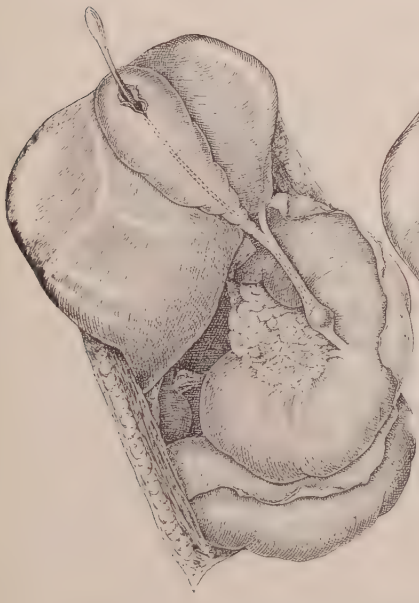

FIG. 41.—RETRODUODENAL CHOLEDOCHOTOMY.

The presence of a stone impacted in the common duct having been determined by a probe passed from the gall-bladder or from an incision in the supraduodenal choledochus, the duodenum is mobilized, and the retroduodenal choledochus exposed.

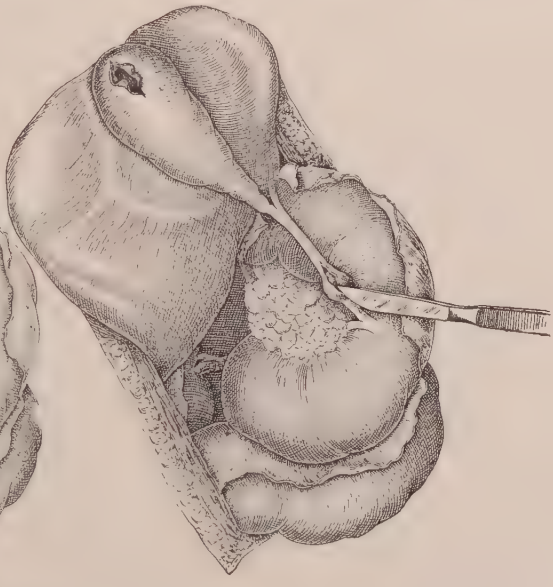

FIG. 42.—RETRODUODENAL CHOLEDOCHOTOMY: THE CHOLEDOCHUS IS INCISED OVER THE IMPACTED CALCULUS.

method employed by Finney (1902) as an aid in his operation of pyloroplasty; and was systematized by Kocher in 1903, in connection with his operation of lateral gastro-duodenostomy (1892) and his method of partial gastrectomy. (See Vol. I, pages 367 and 393).

Operation.—The operation is performed thus: after exposing the parts through the usual incision, the descending duodenum is identified. An incision of 3 or 4 inches long is then made through

the fold of peritoneum just on its right (hepato-duodenal ligament). Into this incision the surgeon inserts his gloved fingers, covered with gauze, and gently wipes the duodenum off the posterior structures, turning it toward the patient's left. As the peritoneal margins are now kept under tension, it is easy to extend the incision upward into the free border of the gastro-hepatic omentum exposing the common bile-duct at the first portion of the duodenum.

FIG. 43.—RETRODUODENAL CHOLEDOCHOTOMY: AFTER REMOVAL OF THE STONE, THE HEPATIC DUCT IS EXPLORED BY PROBE.

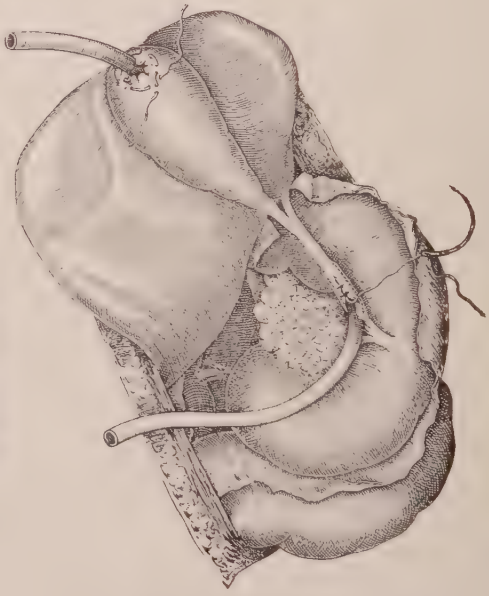

FIG. 44.—RETRODUODENAL CHOLEDOCHOTOMY: DRAINAGE OF THE CHOLEDOCHUS AND OF THE GALL-BLADDER BY SEPARATE TUBES.

From this point the choledochus may now be traced downward. It is found to lie either in a groove in the head of the pancreas or to be completely buried in its substance. In the latter case Vautrin recommends that it be exposed by burning through the overlying pancreatic tissue with the actual cautery. Usually it is sufficient to expose the choledochus by blunt dissection. Bleeding from the lacerated pancreatic tissue may be checked by gauze pressure, or if persistent by suture. The calculus, for the removal of which the operation is done, usually is palpable through the pancreatic tissue and serves as a guide to the exposure of the duct.

The duct is to be incised directly over the stone, which is then extracted through the opening in the duct; its entire lumen is next carefully explored by sound, or by finger if the duct is large enough. All obstructions having been removed, the opening in the duct is closed with through and through catgut sutures (or the sutures may include only the muscular and fibrous coats), and the duodenum is replaced in its normal position, the retroduodenal space being drained for two or three days by a rubber tube or rubber dam, to guard against possible leakage from the choledochus. The abdominal wound is then closed in the usual manner.

Cholecystenterostomy.—This is an operation for the establishment of a fistula between the gall-bladder and a portion of the intestinal tract. The duodenum should be selected for the anastomosis when possible; such an operation is termed a *cholecystoduodenostomy*. If the jejunum is used, it is called *cholecystojejunostomy*. Anastomosis with the colon is known as a *cholecysto-colostomy*. We have already (page 130) expressed our preference for an anastomosis with the stomach, whenever the duodenum is not accessible. The indications and contraindications to cholecystenterostomy have also been discussed at pages 342 and 366.

The anastomosis may be made by direct suture, or by means of a small Murphy button or a Robson bobbin. These methods slightly shorten the time of operation and are more easily performed than the direct suture method, but we prefer the latter because we feel more sure of the firmness of the resulting union of the two viscera. The union when the button is used is very slight immediately following the sloughing of the tissues crushed between the two portions of the button and, as stated by Robson, it is necessary to keep the patient absolutely quiet for two weeks before one can rest assured that the union will not break down and allow escape of the visceral contents.

The abdomen is opened in the usual manner for exposure of the biliary passages, and the operative field within the abdomen is cleared of viscera and walled off as previously described. The gall-bladder is opened at its fundus, and is emptied of calculi, bile or débris. The use of a small pair of Roosevelt anastomosis forceps with rubber-covered blades considerably facilitates the insertion of the sutures, but it is not always possible to free the

structures sufficiently for their application, even when one adopts the method of mobilizing the duodenum, already described (page 466). Whenever possible, a lateral anastomosis is made by suture, as detailed in Vol. I, page 377. The opening in the duodenum is about 1 inch in length, corresponding in size to that made in the gall-bladder. The anastomosis should be carefully inspected for

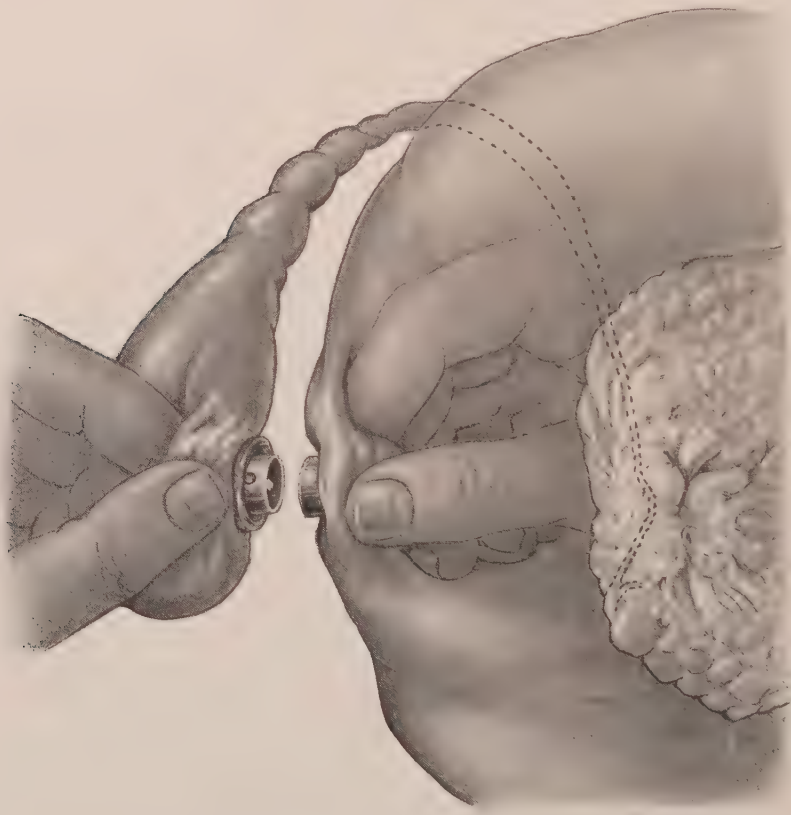

Fig. 45.—Cholecysto-duodenostomy by Means of a Murphy Button.

signs of leakage. The abdominal wound may then be closed without drainage.

When the Murphy button is used instead of the suture method, a small sized button is selected. The opening in the gall-bladder is encircled by a running suture of linen. The portion of duodenum or stomach to be used in the anastomosis is freed of its

contents by gentle stroking, and clamps with rubber-covered blades then are applied for the purpose of preventing any discharge from the gut during the succeeding steps of the operation. A circular suture of linen thread is then run around the site of the proposed opening in the bowel, and the latter is incised just enough to permit the introduction of one end of the button. The suture is drawn tight around the central barrel of the button, and the second part of the button is fastened into the opening in the gall-bladder in a similar manner. The two ends of the button are approximated and pushed together firmly. Then it is well to apply a few interrupted sero-serous sutures to hold the viscera in apposition, and relieve the strain on the anastomosis. This completes the operation, and the abdominal wound may be closed without drainage.

Other methods of biliary intestinal anastomosis, such as **choledocho-enterostomy, hepatico-enterostomy,** etc., have been discussed in Chapter II, page 127.

Operations on the Liver.

Hepatopexy.—This is an operation to fix a movable liver. It is very seldom required. Fixation is best secured by attaching the anterior margin of the liver, particularly the right lobe, to the costal margin by means of heavy sutures of chromic catgut. The greater the number of the sutures, the less will be the strain on any one of them.

Operation.—The abdomen is opened by the incision usually employed for operations on the biliary tract, and the proptosed liver is replaced in its normal position. If it is irreducible, it should be fixed in the best obtainable position by means of sutures passed through the muscular and aponeurotic structures of the anterior abdominal wall. When it has been returned to its normal site, the skin and subcutaneous tissues should be dissected back over the costal margin until this is fully exposed. Then a strong round needle, with large curve, and armed with No. 2 or No. 3 chromic catgut, should be passed from above through the costal margin, between the costal cartilages, directly through the costophrenic structures, emerging in the peritoneal cavity close to the anterior margin of the liver. This is then penetrated from

above downward, and when the needle has been withdrawn from the under surface of the liver, it is at once carried back again to a point about half a centimetre distant from its original place of entrance, thus completing one mattress suture. From two to eight such sutures should be passed. Injury of the pleura has occurred but has caused no harm. The knots of the sutures thus are placed on the superficial surface of the thorax, just beneath the subcutaneous fat. All the sutures should be placed before any one of them is tied; and just before they are tied the convex surface of the liver and the under surface of the diaphragm should be abraded with gauze so as to promote adhesion. The abdominal incision is closed without drainage. The patient should remain recumbent for three weeks.

Fig. 46.—Abscess of the Liver, showing Method of Approach Across Costo-phrenic Sinus.

Depage's operation includes, in addition to hepatopexy by a modified technique, also a rather extensive excision of the abdominal wall (*laparectomy*), as he regards laxity of the abdominal wall as one of the main causes of visceral prolapse. The excision involves the whole of the linea alba, and a more extensive area of the overlying skin and fat. By careful suturing of the anterior and posterior sheaths, the two recti muscles (formerly widely separated by the thinned and stretched linea alba) are brought together in the median line, and the umbilical and falciform ligaments of the liver are drawn taut and fixed in the upper angle of the wound.

Other methods of hepatopexy have been referred to at page 163.

Transpleural Hepatotomy for Abscess of the Liver.—A four-inch subperiosteal resection of the eighth, ninth or tenth rib (according to the site of the abscess) is done, in the mid-axillary line. It is better to excise a rib that is too low than one that is

too high (Fig. 46). If it is thought necessary to remove also a portion of the next rib above, this may be done at the same time, or later. Both ribs may be exposed by an incision over the intervening intercostal space; or, if preferred, a flap of the soft parts may be

Fig. 47.—Transpleural Operation for Hepatic Abscess; Sutures Passing through Deep Layer of Costal Periosteum, both Layers of Unopened Pleura, and Underlying Diaphragm.

turned up, exposing two or three ribs. To avoid infection of the pleura the surgeon should next insert, along the lower border of the highest rib exposed, three or four interrupted sutures of chromicized catgut which include the deep layer of the periosteum,

Fig. 48.—Transpleural Operation for Hepatic Abscess: incision Across Costo-phrenic Sinus.

both layers (parietal and diaphragmatic) of the *unopened* pleura, and the diaphragm (Fig. 47). This plan, employed by W. W. Ashhurst, and advocated by Körte, is much safer than trusting to inward pressure to hold the pleura in contact with the diaphragm,

or than relying on clamps for the same purpose, or even than suturing the two layers of the pleura together after the pleura has been opened, as was orignially done by Knowsley Thornton. It is easy to tell by the sense of touch when the needle catches in the diaphragm. It is unnecessary to insert these sutures anywhere except as advised, *above* the site proposed for the incision through the layers of the pleura and the diaphragm. According to Körte, Roux recommends that these sutures be inserted so that each one interlocks with the next. Having thus shut off the pleural cavity, a 3-inch incision is then made along the upper border of the lowest rib exposed (Fig. 48). This incision divides the deep layer of the periosteum, and both layers of the pleura in the costophrenic sinus. The diaphragm is next incised; this may be done in the same direction as the former incision, or at right angles to it, splitting the diaphragmatic fibres parallel to their course. The right lobe of the liver may now be freely palpated. The liver, in acute cases, is bluish, soft and pulpy, and often bulges into the wound. The region of the abscess is more resistant to palpation, and usually is indicated by denser adhesions. Gauze should be packed around the site proposed for the incision into the liver, and if the finger does not break into the abscess cavity, an exploring needle or grooved director should be pushed into the liver. If more room is needed another rib (the next higher) may be excised, but it is not necessary to enlarge the opening in the pleura. When pus is located, an incision into the abscess cavity should be made along the needle or director as guide. For this incision a scalpel is preferable to the actual cautery advised by some surgeons. A free opening for drainage should be secured by dilating the incision with the finger or dressing forceps. Search should be made for neighboring abscesses, which should be opened through the walls of the first abscess incised.

Drainage is to be secured by placing two large rubber tubes into the abscess cavity. The remainder of the wound is then tamponed with gauze.

According to Mayo, the mortality of the operation is about 30 per cent.; in cases of solitary abscess McCaw places the present mortality as low as 14 per cent.

After-treatment.—Irrigation should not be practised for four or five days at the least. It is facilitated by the use of a double

tube for drainage, as already advised. These tubes may be shortened from day to day, but not too rapidly. The sinus may take many weeks to close.

Appendicostomy may be necessary if the colitis persists.

Hepatotomy for hydatid cysts has been described at page 198.

Hepatorrhaphy, or suture of the liver, is described below, in connection with hepatectomy.

Hepatectomy.—In removing portions of the liver the main problem is *control of hemorrhage during the operation.* The method proposed by Pringle (mentioned at page 242), which consists in clamping the pedicle of the liver, has not been adopted in the human subject, so far as we know. McDill thinks the forceps could be kept in place for fifteen or twenty minutes without doing permanent damage.

If the tumor to be excised possesses a small pedicle, which does not itself require excision, this may be *clamped by crushing forceps.* Kocher prefers the use of very heavy crushing forceps which will squeeze all the parenchymatous tissue away from the line of compression, leaving only the capsule of Glisson, which may be easily sutured.

In some cases it will be possible to secure sufficient hæmostasis by *compressing the liver with the hands* while the tumor is being cut away. In most cases, however, it is more convenient to resort to *elastic constriction* by means of a rubber band tied around the base of the tumor. If this rubber band tends to slip off (as it usually does, unless there is a distinct pedicle) it may be held in place by transfixing the liver with pins, in a method similar to that employed by Wyeth for shoulder- and hip-joint amputations. If nothing else is available, fairly efficient hæmostasis may be secured by inserting a series of *interlocking mattress sutures* all around the area to be removed; if these are tied over some resisting substance, as suggested below, they will not be apt to tear out.

Keen, and some surgeons since his time, employed the *actual cautery* to divide the liver tissue, thus providing for a certain measure of hæmostasis at the same time. If the cautery is used at a dull red heat, and if the section is made very slowly, it is possible to recognize and ligate each large vascular trunk as it is encountered, while the bleeding from the smaller arteries and

veins is checked by the heat. It is possible to catch the larger vessels on forceps, and to draw them out from the parenchyma sufficiently far for the application of ligatures; or a ligature may be passed by means of a curved needle.

Whatever the method of temporary hæmostasis adopted, the section of the liver should be made in wedge shape, so as to facilitate closure of the wound (Fig. 49).

When the tumor has been removed, the sectioned area requires treatment, for the permanent arrest of hemorrhage and oozing of bile. Ligation of the larger individual vessels, already mentioned, should be done whenever possible. *Suture of the*

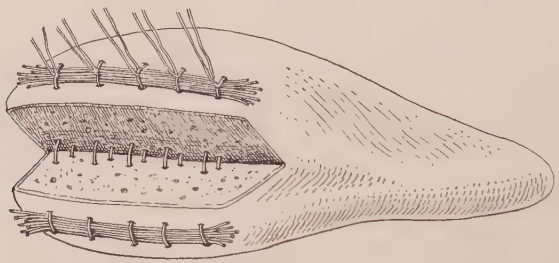

Fig. 49.—Suture of Wedge-shaped Wound of Liver, Left after Resection of a Tumor. The Mattress Sutures are Tied over Strands of Catgut to Prevent Them from cutting out.

liver is difficult only because the usual suture material as ordinarily applied is prone to tear out. The needles employed should be long, rather blunt pointed, and without any cutting edge. For experimental work J. E. Sweet has devised a needle with a female thread at one end, instead of an eye; dry catgut is screwed into this. This plan avoids the "shoulder" formed where the catgut is doubled to pass through the eye of the ordinary needle. Sweet thinks this needle minimizes the traumatism to the liver. Heavy chromicized catgut (No. 2 or 3) is the best material for the sutures, which should be introduced as mattress sutures. They should be tied over a number of strands of catgut, or a strip of fascia, cut from the abdominal aponeurosis, according to the principle of the old fashioned "quilled suture" (Fig. 49). This plan prevents the sutures from cutting out when they are pulled tight, and makes them efficient in checking hemorrhage. Finally the

free margins of the liver flaps should be united with a continuous suture of chromic catgut No. 1.

In all cases drainage is advisable to provide for leakage of blood or bile.

Operations on the Pancreas.

Pancreatotomy for cysts, etc., has been described at page 380.

Pancreatostomy, according to the method of Link, is described at page 349.

Pancreatectomy.—We have already discussed **resection** of the body of the pancreas, and **excision** of its tail (page 365); and it remains for us here to describe the technique employed for excision of the head of the pancreas, or **total cephalic pancreatectomy,** as it is termed by Sauvé.

The chief indications are carcinoma of the head of the pancreas or of the ampulla of Vater invading the pancreas. As extension to the duodenum from the pancreas, and to the pancreas from the papilla of Vater or elsewhere in the duodenum occurs very early, the complete removal of a tumor in either situation usually involves *pyloro-duodeno-pancreatectomy.* The duodenum, it is true, may be removed without impairing the blood-supply of the pancreas, as pointed out by Cotte and Maurizot; but the reverse is not true. Removal of the head of the pancreas requires also removal of the surrounding duodenum, because the blood-supply of the latter is necessarily destroyed.

Early diagnosis is necessary to render such an operation of any value; but as noted at page 362 the classical signs of cancer of the pancreas usually are evidences of inoperable growths.

Operation.—Very free exposure is required. The incision recommended by Desjardins is shown in Fig. 50. The incision of Czerny probably would do as well (page 443).

According to the technique elaborated by Sauvé the operation comprises the following steps: (1) Abdominal incision. (2) Ligation of the pyloric and gastro-duodenal arteries and section of the pylorus. (3) Mobilization of the duodenum. (4) Section of the transverse duodenum, just to the right of the superior mesenteric vessels. (5) Separation of the so-called "little pancreas" from beneath the superior mesenteric vessels, and of the head of the pan-

creas from the portal vein. (6) Section of the head from the tail of the pancreas; second ligation of the gastro-duodenal artery; ligation and section of the choledochus.

These steps terminate the operation of duodeno-pancreatectomy proper, but there still remain to be done the supplementary operations of gastro-enterostomy and cholecystenterostomy or some of their modifications. Finally, some disposition must be made of the sectioned tail of the pancreas. Sauvé advises fixing this in the abdominal wound, by a method of pancreatostomy somewhat analogous to that employed by Link (page 349). Desjardins suggested that it be implanted into a loop of jejunum; and by means of Coffey's proposed technique for pancreato-enterostomy, which was successful in dogs, this disposition of the pancreatic stump might be safely accomplished in life.

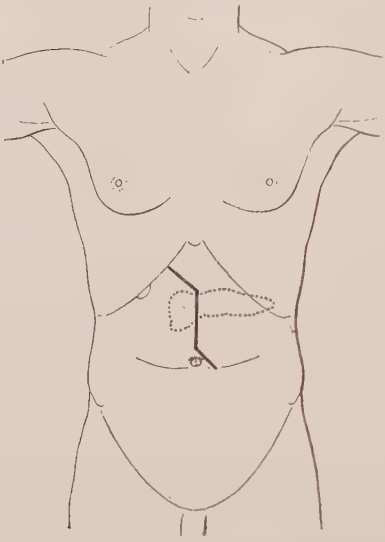

Fig. 50.—Incision for Total Cephalic Pancreatectomy. (*Desjardins.*)

This operation of duodeno-pancreatectomy is so extensive and requires so long a time for its performance that it probably would be better to perform it in two stages. As the most urgent symptom usually is the presence of obstructive jaundice, the first operation might be planned to relieve this by some form of biliary and intestinal anastomosis; but the extremely unfavorable prognosis which attends such operations for malignant obstruction renders their employment hazardous in the extreme. It seems probable, therefore, that the radical operations for carcinoma of the head of the pancreas could be indicated only when the condition was found unexpectedly when operating upon a fairly healthy patient. Under such circumstances it would be proper, we believe, to perform at the first operation *posterior gastro-jejunostomy and exclusion of the pylorus* (Vol. I, page 384); at the second operation, two weeks or more later, duodeno-pancreatectomy may be attempted, and in cases of emergency the bile and pancreatic juice might be

drained externally for a time, and the supplementary operations of cholecyst- (choledocho-) enterostomy and pancreato-enterostomy be postponed to a third intervention.

Though such extensive operations on patients so gravely ill seem to offer little prospect even of immediate survival, and still

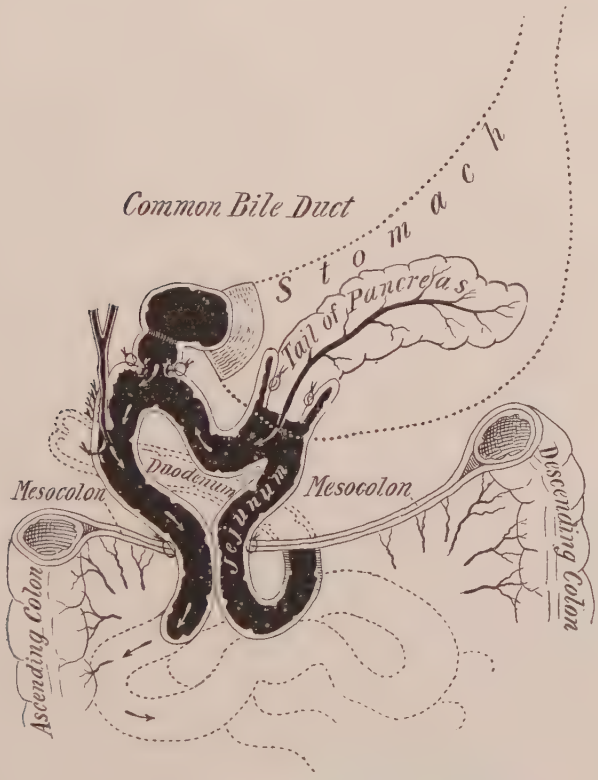

FIG. 51.—PANCREATO-ENTEROSTOMY. (*Coffey.*)

After duodeno-pancreatectomy, the body of the pancreas is implanted into the apex of a loop of jejunum. The jejunum is anastomosed with the pylorus, and the choledochus is implanted in it just distal to this anastomosis.

less of ultimate cure, it should not be overlooked, as Sauvé has pointed out, that even in unpremediated pancreatectomies, done without a well-ordered technique, and with no previous experience, the successes have been more numerous than the failures (page 366). Sauvé concluded that when the surgeon should become possessed of a carefully ordered technique for total excision of the head

of the pancreas, and when he should have clearly recognized the indications for the operation, that then the successes would be more numerous, and above all more lasting.

Pancreato-enterostomy.—Desjardins, as already mentioned, proposed to implant the sectioned end of the pancreas into the intestinal canal. The technique of the operation has been worked out by Coffey in experiments on dogs. He found end-to-end anas-

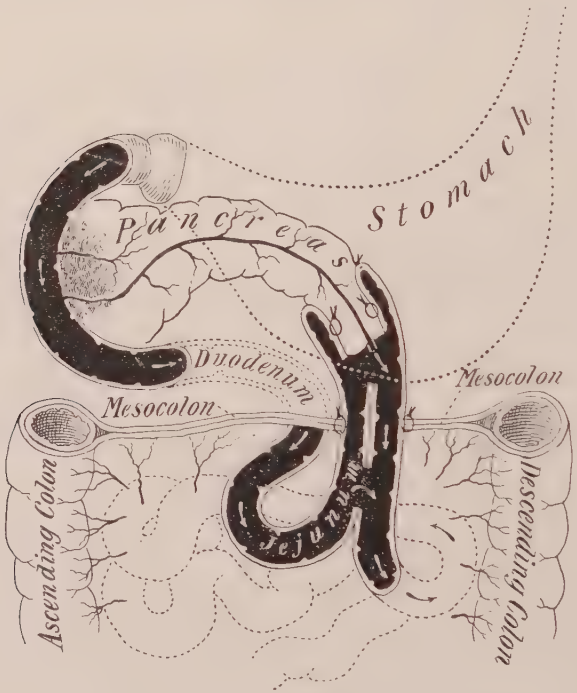

FIG. 52.—PANCREATO-ENTEROSTOMY. (*Coffey.*)
As a palliative operation the tail of the pancreas may be implanted into the apex of a jejunal loop.

tomosis was preferable to end-to-side implantation. After section of the jejunum, the continuity of the intestinal tract is restored by implanting the upper jejunal loop into the side of the lower (Y-anastomosis); the open end of the aboral segment of jejunum is then employed for the reception of the sectioned pancreas. Coffey found, however, that the lumen of the bowel was not sufficiently capacious to permit of the intestinal wall being inverted around the stump of the pancreas after the latter had been

introduced into the bowel. He therefore adopted the method of throwing the lumina of two intestines into one by making his opening in the bowel at the apex of a loop (Fig. 52). In this way it was possible to obtain a very secure anastomosis.

Operations on the Spleen.

Splenectomy.

Incision.—In traumatic cases the best incision is one above the umbilicus to the left of the median line, as it enables the operator to examine the other viscera. If difficulty is experienced in exposing a spleen which is fixed by adhesions, the left rectus muscle should be divided transversely, as in Czerny's similar incision on the right side for difficult operations on the bile-ducts. In other cases, an incision through the outer border of the left rectus, continued, if necessary, up the rib margin to the ensiform cartilage gives readiest access to the spleen. This is similar to Mayo Robson's incision on the patient's right, for operations on the biliary tract. Sometimes it is easier to reach the pedicle of the spleen from the inner side through a median incision, but usually an incision through the left rectus near its outer border is the most satisfactory.

The abdomen having been opened, the future steps of the operation depend on the presence or absence of adhesions. The latter usually are most numerous between the diaphragm and the upper pole of the spleen although omental adhesions are not uncommon. Before proceeding further, the splenic region should be isolated by gauze pads.

The most important part of the whole operation is the *control of hemorrhage*. The capsule of the spleen is easily torn and the pulp is soft and friable. The walls of the veins in the pedicle are thin and tear readily or else are calcareous and brittle. Gentle manipulation therefore is necessary.

It is best to work from below upwards, clamping the vessels before cutting them. Adhesions should be cut between clamps or ligatures. The vessels in the pedicle can be more easily reached if the spleen is turned over, but this is not possible until the adhesions are broken down, and the gastrosplenic and lienophrenic folds of peritoneum divided. If the hand can be introduced gently between the spleen

and diaphragm, the former may be drawn down into the wound. It then can be turned over, and the vessels in the pedicle may be isolated by blunt dissection. If they are not too large they may be secured in two long clamps which should be applied as close as possible to the spleen; the pedicle may then be cut between them and the spleen. In three out of thirty-one splenectomies, Mayo injured the pancreas, and had to include its stump in his ligatures. All three patients recovered. There must be enough tissue left in the pedicle to admit of the safe application of ligatures. When the bleeding has been stopped the isolating packs are removed and the abdominal incision is closed in layers in the usual manner. After splenectomy for abscess, or when there is some oozing from adhesions, or in traumatic cases, it may be advisable to leave some gauze drainage in the wound. It can be brought out at the lower angle of the incision or through a stab wound below the rib margin.

Splenotomy.—Cysts and abscesses are the indications for splenotomy and as a rule it is undertaken because splenectomy is difficult or impossible owing to adhesions. Under such circumstances the general peritoneal cavity is rather well walled off by the adhesions, but in any case, if exposed, it should be carefully protected with gauze before the spleen is cut into. The abdominal incision may be made over the most prominent portion of the tumor or through the semilunar line. There are no special points in technique.

Abscesses and cysts that develop backward often can be reached more easily by a transpleural operation, resecting a portion of the ninth or tenth rib in the postaxillary line, sewing the layers of the pleura together to shut off the lung and general pleural cavity, and then going through the diaphragm into the abdomen.

The spleen may also be reached through the lumbar incision for kidney operations, if this is extended well forward. On account of mistaken diagnosis this has been done quite frequently.

Splenopexy.—Splenopexy is an operation that is very seldom performed. It is indicated in comparatively few cases, such as those with the spleen normal in size but freely movable, and without any pathological change in its substance or its capsule. These cases occur very rarely.

Moynihan mentions various methods. 1. Suture of the spleen to the diaphragm or abdominal wall (Tuffier). This is very likely to cause severe hemorrhage. 2. Fixing the spleen in place by surrounding it with gauze to excite the formation of adhesions (Kouwer). 3. Burying the spleen beneath a pocket of peritoneum on the diaphragm (Rydygier). 4. Bringing the spleen out of the abdomen into the lumbar fossa and fastening it there to make it a retroperitoneal organ (Bardenheuer). 5. A case described by Basil Hall in which the lower pole of the spleen was fastened between the layers of the abdominal wound.

Of all these methods practically the only one that is used now is that of packing gauze around the spleen so that it will be held in its normal position by the formation of adhesions.

No fatalities are reported after splenopexy. In most cases the result has been satisfactory, but, as mentioned at page 400, splenectomy was required later in a patient in the German Hospital, owing to increase in size of the spleen.

REFERENCES.

Ashhurst, W. W.: Amer. J. Med. Sc., 1905, i, 140.
Assmy: Beitr. z. klin. Chir., 1899, xxiii, 109.
Bevan: Annals of Surgery, 1899, xxx, 17.
Coffey: Annals of Surgery, 1909, l, 1238.
Collins: Surg., Gyn. and Obst., 1908, vi, 413; 1909, viii, 293.
Cotte and Maurizot: Lyon Chir., 1910, iii, 629.
Courvoisier: Beitr. z. Path. u. Chir. d. Leber u. Gallenwege, Berlin, 1890.
Czerny: Deutsch. med. Woch., 1892, xviii, 518.
Depage: Arch. f. klin. Chir., lxiii, 1081.
Desjardins: Thèse de Paris, 1905 (Choledochotomy, p. 110).
Desjardins: Rev. de Chir., 1907, xxxv (Pancreatectomy).
Duval: Thèse de Paris, 1902.
Elliot: Annals of Surgery, 1895, xxii, 97.
Gosset: Bull. Soc. Chir., Paris, 1912, xxxviii, 1174.
Jourdan: Thèse de Paris, 1895. "De la Choledochotomie," p. 153.
Kausch: Handb. d. prakt. Chir. (v. Bergmann, Mikulicz, et al.), Stuttgart, 1900, Bd. iii; i Theil, S. 128.
Kausch: Beitr. z. klin. Chir., 1910, lxxi, 691.
Kehr: Technik d. Gallensteinoperationen, München, 1905, S. 64.
Kehr: Arch f. klin. Chir., 1912, xcvii, 74 (New incision).
Kocher: Corr.-Bl. f. Schweizer Aerzte, 1895, xxv, 193 (Internal choledochoduodenostomy).
Kocher: Zentralbl. f. Chir., 1903, xxx, 33 (Mobilization of duodenum).

Kocher: Operative Surgery, English translation by H. J. Stiles, London, 1911 (Abdominal incision, p. 534; cholecystendysis p. 538).
Körte: Chir. Operationslehre (Bier, Braun, Kümmell), Leipzig, 1912, Bd. ii, S. 711 (Has abandoned his oblique incision for Sprengel's).
Langenbuch: Verh. d. Deutsch. Gesellsch. f. Chir., 1896, xxv, ii, 68.
Leriche: Revue de Chir., 1906, xxxiv, 110.
Mayo: Keen's Surgery, Phila., 1908, vol. iii, p. 975.
Mayo: Trans. Amer. Surg. Assoc., 1913, xxxi.
McCaw: Keen's Surgery, Phila., 1909, vol. iv, p. 1114.
McDill: Trans. Amer. Surg. Assoc., 1912, xxx, 106.
Meredith: Brit. Med. Jour., 1885, i, 431 (Cholecystendysis in 1883; death next day).
Moynihan: Keen's Surgery, Phila., 1908, vol. iii, p. 1092.
Nussbaum: Die Verletzungen des Unterleibes (Deutsche Chir., Lief. 44), Stuttgart 1880, S. 94.
Ochsner: Surg., Gyn. and Obstetr., 1906, ii, 204.
Quénu and Duval: Revue de Chir., 1905, xxxii, 401 (Retrograde catheterism of bile-ducts).
Robson: Diseases of Gall-bladder and Bile-ducts. 3d Ed., London, 1904, p. 263 (Abdominal incision).
Robson and Cammidge: Gall-stones, their Complications and Treatment, Oxford University Press, 1909.
Sauvé: Rev. de Chir., 1908, xxxvii, 113; 335.
Sick: Beitr. z. klin. Chir., 1910, lxxi, 691.
Sprengel: Verhandl. d. Deutsch. Gesell. f. Chir., 1910, xxxiv, ii, 132–136.
Tait: Lancet, 1879, ii, 730.
Terrier: Bull. Soc. Chir., Paris, 1905, xxxi, 1101.
Thornton: Brit. Med. Jour., 1886, ii, 903.
Vautrin: Revue de Chir., 1896, xvi, 446.
Wiart: Thèse de Paris, 1899 ("Anatomie Topographique et Voies d'Accès du Cholédoque").
Zeller: Berl. klin. Woch., 1902, xxxix, 822 (Retrograde catheterism of bile-ducts).

INDEX OF NAMES

Abbe, 129
Abrami, 260
Adami, 5, 18
Addis, 32
Alberti, 254
Albu, 160
Alexander, E. G., 319
Allessandri, 140
Apolant, 139
Archibald, 3, 328, 338
Aristotle, 254
Arnaud, 282, 299, 300, 307
Arnsperger, 266, 317
Aschoff, 11, 15, 50, 75, 217
Asellius, 254
Ashhurst, W. W., 379, 473
Assmy, 441
Auvray, 181, 215 221, 252,

Bacmeister, 8, 9, 11, 15, 50, 75, 217
Balser, 255, 280
Band, 258
Banti, 408, 409, 413, 418, 420, 430
Bard, 356
Bardeleben, 131, 211
Bardenheuer, 483
Barker, 337
Barnard, 152
Baron, 242
Barr, 409
Barrier, 192
Bartels, 315
Bartlett, W., 14
Bashford, 354
Battersby, 278
Baudouin, 128
Bauhinus, 254
Beadles, 90, 213, 218, 219
Becker, 351
Benczur, 351
Bender, 281
Benvenute, 29
Berg, 165
Berger, 128, 129
Bernard, 279
Bernheim, 33
Bernouilli, 337
Besredka, 33
Bier, 128
Billroth, 365
Biondi, 366
Bircher, 305
Bishop, 5, 6, 8, 10,
Bloom, Homer C., 174
Böcker, 189
Bode, 140
Bogojawlensky, 186
Böhm, 281, 284, 291, 353
Boinet, 190
Boldt, 283
Boljarski, 235, 243, 244
Bond, 2, 4, 199
Bosanquet, 270
Bovaird, 411
Bramwell, Byron, 259
Brandt, 133
Bremser, 189
Brentano, 130, 302, 308, 309
Brewer, 132, 136, 309
Brewitt, 291
Brin, 134
Brissaud, 207
Brockbank, 88, 90, 151, 153
Brown, T. R., 161
Bryan, 422
Bryant, 281
Bubenhofer, A., 41
Budd, 66
Bunge, 187, 281, 301, 304
Burckhardt, 235
Burghard, 409
Busch, 290, 291

Camac, 22
Cammidge, 101, 257, 271, 278, 282, 292 336, 369
Cantani, 160
Cantlie, 164
Capparelli, 278
Capriani, 348
Carnot, 265, 278, 289, 337
Cartier, 337
Cauchoix, 192, 198, 199
Cawley, 270
Cecil, 367
Chauffard, 2, 332, 334, 337, 357
Cheyne, 160, 163
Chiari, 241, 278, 290, 294
Chittenden, 389
Chopart, 255
Chvostek, 283
Citernesi, 420
Clairmont, 54
Clark, 159
Clarke, 160
Clayton, 269
Clegg, 171
Codivilla, 366
Coe, 86
Coenen, 305, 308
Coffey, 366, 478, 480

Coffin, 168, 180
Collins, 442
Collinson, 53
Combenale, 185
Connelly, 369
Cordier, 123
Cotte, 121, 477
Courvoisier, 76, 83, 128, 144, 218, 223 251, 447
Cranwell, 198, 201
Crile, 32
Crowe, 62
Cruveilhier, 254
Cumston, 208
Cunéo, 366
Cushing, 206
Cyr, 175
Czerny, 127, 131, 134, 138, 224

Dald, 135
Davaine, 189
Davis, G. G., 54
Davis, W. E. B., 129
Deaver, H. C., 66
Dehler, 27
Delagenière, 185
Delbet, 135, 199
Desjardins, 264, 269, 289, 309, 317, 334, 366, 462
Deucher, 277
Dévé, 193, 199
Dick, 300
Dieckhoff, 254
Diehl, 351
Dieulafoy, 179, 207, 290
Doberauer, 133, 282
Doberer, 138
Dock, 187
Dolley, 159
Donati, 221
Donhauser, 387
Doran, 53
Dörr, 5
Douglas, 30, 31, 86, 88, 189
Downs, 206
Doyen, 133
Dreesman, 299, 337
Dubar, 185
Durham, 153
Dutil, 353
Duval, 201, 284, 366, 467

Eastman, 91
Ebner, 266, 281, 300, 305
Ecklin, 360
Edgecomb, 261
Edler, 235, 240, 251
Edwards, 153
Egdahl, 262, 284, 292
Ehrhardt, 138
Eichmeyer, 130
v. Eiselsberg, 272
Elösser, 301
Elliot, 296, 441
Else, 6, 19
Enderlen, 127, 135
Engelhardt, 207
Eppinger, 29, 45, 353
Erdman, 53, 337
Eshner, 41
Estes, 72, 258
Eudemus, 254
Ewald, 171
Exner, 8

Fallopius, 254
Fasano, 308
Faure, 160
Fedeli, 277, 336
Fenger, 78, 461
Ferguson, 353
Fertig, 241
Fetzer, 212
Ficai, 420
Fiedler, 88
Filippini, 216
Fink, F., 41
Finney, 258, 365, 367, 467
Finsen, 194
Finsterer, 235, 238
Fischer, 55
Fitz, 255, 260, 276, 288, 335
Fles, 276
Flexner, 175, 265
Flint, 8
Florcken, H., 90
Foote, E. M., 53
Forster, 27
Fournier, 2
Föwelin, 351
Fowler, R. S., 192
Fowler, 199
Frank, J. P., 86
Franke, 163, 315, 366
Frankel, 2
Frazier, C. H., 56
Freeman, 215
Frerichs, 14
Freytag, 388
Fullerton, 134
Funk, 2
Funke, 91
Fütterer, 218

Galen, 189, 254
Galippe, 268
Galliard, 269
Garrè, 74, 138, 141
Gaultier, 275, 286
Gay, 41
Gérard, 9, 163
Gerhardt, 29
Gersuny, 53, 130
Ghedini, 197
Gibbon, 54, 59
Giffin, 435

Gilbert, 2
Giordano, 236
Giudiceandrea, 268, 278
Glénard, 161
Glinski, 257
Goodall, 355
Goodman, 279, 337
Gordinier, 206
Gorstell, 90
Gosset, 444
Graff, 268, 418, 433
Graham, 160
Graser, 241
Greenough, 187
Gregg, 26
Griffith, 40
Grimme, 27
Gross, 308, 335
Grunert, 140
Guibert, 258
Guinard, 294, 308, 309
Gulecke, 281
Gussenbauer, 255
Gutteridge, 149

Haasler, 119, 129
Haberer, 54
Habs, 140
Hagen, 337
Hahn, 292, 300, 305
Hall, Basil, 483
Haller, 86, 254
Halsted, 224, 300
Hamilton, Edward, 188
Hammond, 26
Hansemann, 282
Harley, 180
Harris, 159, 261, 409
Harte, R. H., 242
Hartmann, 85, 115, 134
Heiberg, 335
Heidenhain, 28
Hein, 8
Heineke, 387
Heister, 160
Heller, 140
Helly, 267, 280
Helm, 258
Henzelman, 237
Herophilus, 254
Herrick, 168, 180
Hertz, 157
Herzen, 389
Herzog, 238, 409
Hess, 40, 241
Heurnius, 254
Heusner, 128
Hewlett, 280, 337
Heyrovsky, 8
Hildebrandt, 129
Hill, 414, 430
Hillier, 355
Hippocrates, 189
Hirschberg, 129
Hirschfeld, 262
Hochenegg, 215
Hoffman, Friedr., 86
Holscher, 165
Holzmann, 278
Homans, 12
Hoppe-Seyler, 2, 12, 86
Horton-Smith, 165
Hotchkiss, 60
Houghton, 238
Hubbard, 237, 240, 241, 388
Hulst, 354
Hunter, 34

Israel, 181, 197

Jaboulay, 133
Jacobs, 216
Jacobson, 292
v. Jaksch, 433
Janowski, 218
Jenckel, 136
Jimenez, 169
Johansson, 54
Johnston, 346, 407, 432, 434
Jones, 72
Jourdan, 467
Jundell, 26
Jürgensen, 277
Jurist, 294
Justi, 127

Kahlden, 211
Kakels, 367
Katz, 275
Kausch, 130, 134, 224
Keen, 207, 215, 475
Kehr, 12, 15, 51, 65, 68, 86, 90, 93, 110, 112, 130, 133, 134, 135, 139, 140, 158, 189, 212, 250, 284, 292, 317, 330, 337, 444, 450, 461, 464
Kelly, A. O. J., 2, 3, 11, 15, 18, 34, 50, 63, 87, 157, 158, 164, 182
Kieffer, 168
Kilgour, 249
Kinney, 279
Kinnicut, 337, 348
Kirchheim, 209
Kirschner, 426
Klebs, 371
Klob, 258
Knott, 211
Koch, Joseph, 6, 19
Kocher, 110, 119, 128, 447, 464, 466, 467, 475
Konjetzni, 217, 218
Körte, 128, 131, 133, 142, 224, 258, 282, 291, 300, 302, 333, 337, 375
Koslowsky, 187
Kouwer, 483
Kramer, 8, 10
Krause, 2, 130

Krauss, 90
Krietnitz, 337
Krukenberg, 130
Kübig, 42, 55
Kühn, 120, 353
Kulenkampff, 255
Kümmell, 119, 128, 347
Kuntzmann, 274
Küster, 461

Labbé, 337
Lacher, 258
Laennec, 193
Lameris, 139
Lancereaux, 255, 256, 270
Landau, 161, 198
Langenbuch, 12, 76, 83, 98, 119, 128, 171, 206
Langerhans, 254
Lanphear, 136
Lapointe, 216
Lartigau, 5
Laudenbach, 387
Lauenstein, 461
Lavenson, 39, 40, 79, 272
Lazarus, 268, 346, 372
Leary, 18, 27, 32
Le Bec, 154
Lecène, 300
Lediard, 66
Legg, 160
Leichtenstern, 153
Lejars, 136, 139
Lemoine, 261
Lenormant, 300
Leonardi, 148
Lepine, 337
Leriche, 121, 282, 299, 300, 307, 354, 360 467
Letienne, 2
Letulle, 256, 354
Leue, 159
Lewerenz, 249
Lewis, Morris, 416
Levy, 424
Lichtheim, 348
Licini, 217, 221
Lieutaud, 266
Link, 349, 478
Lippman, 2, 4
Lintvarev, 418
Littlewood, 292
Lloyd, 31, 292
Loewy, 244
Loison, 166
Lorand, 91
Lotsch, 426
Löwi, 272
Lücke, 215, 255
Ludolph, 278
Luschka, 54
Lyle, 276
Lynch, 336
Lyon, 400

McBurney, 101, 107, 120, 464, 466
Macaigne, 260
MacCarty, 117
McCaw, 474
McDill, 178, 475
MacKenzie, 197
McWilliams, 85, 167, 187
Madelung, 189
Manson, 178, 180
Marckwald, 206
Marshall, 241
Martin, F., 156
Martina, 281, 286
Marwedel, 134
Mason, 18
Mastin, C. H., 129
Matheiu, 39, 124–129, 166, 179
Mauclaire, 244, 366
Maugeret, 265, 266, 323
Maurizot, 477
Maury, 131
Mayer, 55
Maylard, 139
Mayo, 32, 35, 59, 89, 112, 115, 128, 133 134, 217, 220, 284, 301, 323, 435, 474, 482
Mayor, O., 258
Meckel, 8
Melchior, 165
Mendel, 389
Mercadé, 240
Meredith, 447
v. Mering, 255, 270
Mettin, 310
Meyer, 12, 387
Michel, 308, 328
Mieczkowski, 2
Mignot, 12, 15, 90
Mikulicz, 140, 444
Millard, 99
Minkowski, 28, 255, 270
Mirallié, 354, 360
Monprofit, 128, 130, 187
Monserrat, 102
Morgagni, 86
Morris, 189
Morris, Henry, 192
Morris, R. T., 314
Moschowitz, 133
Moynihan, 12, 70, 77, 81, 90, 126, 133, 158, 163, 301, 331, 337, 357, 366, 483
Mugnai, 420
Mühsam, 55
Müller, 6, 154, 275
Munro, 167
Murard, 433
Muroya, 266
Murphy, 101
Musgrave, 171
Muspratt, 304
Musser, 217, 218, 260, 285, 292

Naunyn, 3, 8, 9, 12, 15, 28, 66
Niemack, 41
Nimier, 304
Noguchi, 280, 351, 386
Noetzel, 241
v. Noorden, 272
Nötzel, 292, 301, 304

Ochsner, 277, 337, 454
Oehler, 128
Oggi, 3
Olivier, 354
Opie, 256, 262, 264, 276, 279, 317, 323
Oppenheimer, 126, 224
Orsos, 433
Orth, 254
Oser, 353, 354
Osler, 409
Ottenberg, 31, 33
Otto, 15
Outerbridge, 224

Packard, 14
Paget, 249
Pallas, 189
Paracelsus, 8
Parkes, 119
Pascale, 186
Pawlow, 389
Payr, 281
Péan, 163
Peiser, 291
Pellman, 204
Pemberton, 272
Pepper, 348
Perkins, 407
Perls, 212
Peters, 86
Petersen, 126
Petit, J. L., 120
Petry, 63
Phillips, S., 260
Pic, 356
Pilcher, 337
Piorkowsky, 165
v. Pirquet, 32
Pitt, 292
Poncet, 360
Ponfick, 433
Porter, 153, 304
Posadas, 199
Posselt, 89
Power, D'Arcy, 417
Pringle, 242, 475
Pröscher, 218
Prudden, 191
Purser, 42

Queirolo, 29
Quénu, 20, 23, 33, 136, 166, 179, 201, 226, 284
Quincke, 8, 28, 30

Rahn, 258
Railton, 369
Ransohoff, 292
Ravenna, 367
v. Recklinghausen, 204, 255, 262
Regner de Graaf, 254
Rehn, 66
Rentoul, 259
Rheinstein, 102
Rhoads, 168, 175
Richardson, 113, 116, 189, 369, 380
Richet, 260
Richter, 14, 294
Ricketts, 185, 186
Riedel, 55, 66, 93, 127, 134, 140, 255, 285, 461
Ries, 134
Rivet, 334
Roberts, 153, 207
Robinson, Byron, 163
Robson, 46, 66, 76, 80, 101, 131, 135, 151, 215, 250, 255, 257, 270, 278, 279, 282, 292, 301, 310, 313, 323, 329, 337, 354, 357, 369, 446, 461, 464, 469
Roddick, 260
Roger, 277
Rokitansky, 254, 294
Rolleston, 15, 39, 88, 90, 182, 207, 221, 292, 409
Romanelli, 277, 336
Rose, E., 258
Rosenberger, 134
Roser, 197
Ross, 53, 400
Roth, 88
Rother, 86, 88
Rotter, 134, 308, 309
Rouis, 171
Roux, 216, 474
Rowlands, 134
Ruggi, 366
Runge, 258
Russell, 14, 199
Ryan, 199

Sailer, 262, 328
Saint-Girons, 260
Salomon, 274, 277
Santoni, 194
Santorini, 254
Sappey, 258
Sauvé, 224, 365, 477
Sawyer, 206
Schiassi, 420
Schick, 32
Schievelbein, 54
Schiff, 389
Schirmer, 264
Schlecht, 335
Schlössman, 79
Schmackpeffer, 260
Schmidt, 276, 292
Schmorl, 241

Schnitzler, 241
Schröder, 86, 88, 215, 218
Schüller, 221
Schupmann, 269
Schüppel, 14, 79, 207
Schwarz, 31, 33, 337
Segrè, 51, 354, 367
Seidel, 337
Seifert, 9
Selberg, 292
Selten, 433
Sendler, 129, 366
Senn, Nicholas, 255, 370
Serey, 66
Sharp, Gordon, 260
Shattuck, 369
Sheldon, 56, 426
Sherren, 42
Sick, 441
Siegert, 218
Simmons, 207
Simon, 277, 353
Simonds, 414
Simonin, 261
Slajner, 224
Snell, 90
Solieri, 56
Souligoux, 135
Spannaus, 130
Speese, 262, 337
Spiess, 255
Sprengel, 135
Ssaweljew, 160
Stéfani, 367
Stengel, 411
Stich, 294
Stieda, 304
Still, 87
Stone, 41
Stubenrauch, 126
Sullivan, 127, 131
Sutherland, 409
Swain, 135
Sweet, 272, 476

Taft, 337
Tait, 53
Taylor, 337
Tédenat, 135
Terrier, 53, 76, 119, 128, 130, 136, 158, 181, 221, 252, 442, 447, 456, 461, 467
Thévenet, 283
Thévenot, 423
Thiersch, 255
Thiroloix, 266, 370
Thöle, 238, 253
Thomas, 20, 23
Thomson, John, 87, 259
Thompson, 191
Thornton, 14, 120, 128, 181, 185, 199, 474
Tilton, 235
Tinker, 139
Tixier, 260
Tomaschny, 296
Torrance, 411
Trafeyer, 278, 294, 310
Treves, 40, 66
Tricomi, 366
Troisier, 260
Truhart, 263, 282, 289, 291
Tschepurowski, 281
Tuffier, 136, 140, 166, 366

Ulmann, 128
Umber, 418
Upcott, 224

Vautrin, 119, 328, 337, 342, 467
Végas, 198, 201
Verhoogen, 137
Vesalius, 254
Vidal, 184, 258
Vierhuffs, 165
Vierordt, 191
Villandre, 140
Villar, 269, 310
Villard, 283, 337, 367, 433
Villaréal, 366
Virchow, 254, 370
Voelcker, 131, 137
Volhard, 26, 63

Waring, 171
Warthin, 387
Watson, 279, 337
Weeks, 67
Weiss, 79
Weisser, 189
Weissker, 139
Welch, 32, 33
West, 410
White, Hale, 182, 212, 353
Wille, 280
Williams, 241, 290, 291
Wilms, 130, 132, 137
Wilson, 337
Winckler, 433
v. Winiwarter, 129
Winsor, Henry, 153
Winton, 218
Wirsurg, 254
Woh'gemuth, 280, 311, 351, 381
Wolff, 54
Wrzosek, 6, 19

Yeomans, 215

Zancarol, 169
Zenker, 218, 241

INDEX

Abscess, liver, 164
ætiology, 165
amœbic, 168
diagnosis, 178
pain, 175
appendicular, 166
classification, 165
diagnosis, 175
exciting causes, 169
hepatotomy, 472
pathology, 169
post-operative treatment, 474
prognosis, 179
pyæmic, 165
symptoms, 172
traumatic, 165
treatment, 180
tropical, 168
pancreas, 307
operation, 308
treatment, 308
pericholecystic, 71
spleen, 404
diagnosis, 406
pathology, 405
symptoms, 405
treatment, 407
Absence of gall-bladder, congenital, 40
Accessory spleen, 385
Acne pancreatica, 370
Adenoma, liver, 206
symptoms, 207
treatment, 208
pancreas, 367
Adhesions, pericholecystic, 53
Age, in cholelithiasis, 84
Amœba coli, 169
Ampulla of Vater, sphincteric action, 3
Anæmia, splenic, 408
Aneurism, hepatic artery, 140
ligation, 141
in biliary obstruction, 140
Anomalies of liver, 157
Ascites, operations for, 185
Azotorrhœa, in pancreatitis, 276

Bacillus capsulatus aërogenes, 3
choleræ, 3
coli communis, 2
gasoformans, 3
lactis erythrogenes, 3
limbatus, 3
of Friedländer, 3
pyocyaneus, 5
subtilis, 3
typhosus, 2
Bacteriology, Banti's disease, 410
of biliary tract, 2
Bacterium oxygenes, 3
Ball-valve obstruction, 33
Banti's disease, 408
ætiology, 409
bacteriology, 410
diagnosis, 415
pathology, 411
symptoms, 412
treatment, 419
Bayonet incision, 442
Bevan's incision, 445
Bile, absorption, 28
bactericidal power, 2
discharge into duodenum, 19
in urine, 30
stasis, 11
Bile-ducts, anomalies, 39
carcinoma, 217
diagnosis, 222
primary, 221
symptoms, 222
treatment, 223
injuries, 235, 249
kinking, 139
malposition, 39
obliteration, congenital, 39
obstruction, 122
operations, 440
stricture, 123
congential, 39
treatment, 124
Biliary cirrhosis, 182
colic, 95
in pancreatitis, 297
treatment, 117
concretions, *see* Gall-stones.
fistula, 144
external, 148
internal, 144
symptoms, 145
treatment, 148
post-operative, 150
statistics, 144
obstruction by aneurism, 140
external, 139
peritonitis, 53
tract, avenues of infection, 3
common duct, 3
contiguity, 6
lymphatic system, 6
portal circulation, 5
systemic circulation, 5
bacteriology, 2
casts of, 16

Biliary tract infection, 1
retention cyst, 204
surgical diseases, 1
ætiology, 1
pathogenesis, 7
typhoid infection, 17
sequels, 22
Black jaundice, 357
Blood, coagulation time, 31
in jaundice, 31

Calculus, biliary, *see* Gall-stones.
pancreatic, 268, 346
diagnosis, 348
operation, 348
symptoms, 346
treatment, 348
Carcinoma, bile-ducts, 217
diagnosis, 222
primary, 221
symptoms, 222
treatment, 223
gall bladder, 217
pathology, 218
symptoms, 220
treatment, 220
liver, 211
diagnosis, 214
pathology, 212
symptoms, 213
treatment, 215
pancreas, 353
diagnosis, 362
operation, 365
pathology, 354
symptoms, 356
treatment, 365
Cammidge's reaction, 278, 336
Carlsbad cure, 110
Catarrhal jaundice, 45
Cephalic duodeno-pancreatectomy, 366
Cholangeitis, 44
catarrhal, 45
acute, 45
chronic, 47
diagnosis, 47
treatment, 47
suppurative, 47
diagnosis, 49
operation, 49
treatment, 49
Cholangeiostomy for ascites, 158
in cirrhosis, 185
Cholecystitis, 44
acute, 50
catarrhal, 51
diagnosis, 59
differential, 60
operation, 62
prognosis, 61
results, 58
treatment, 61
calculous, 63
Cholecystitis, calculous, treatment, 120
chronic, 62
diagnosis, 64
symptoms, 63
treatment, 64
fever, 57
gangrenous, 55
jaundice in, 58
muscular rigidity, 57
nausea in, 57
non-calculous, 55, 63
pain in, 56
phlegmonous, 54
symptomatology, 56
suppurative, 52
tenderness, 57
typhoidal, 20
operation, 21
primary, 23
cases, 24
ulcerative, 55
vomiting in, 57
Cholecystectomy, 454
for cholelithiasis, 116
indications for, 116
Cholecystendysis, 447
Cholecystenterostomy, 129, 469
in cirrhosis, 185
nature's, 145
statistics, 131
Cholecysto-colostomy, 469
Cholecysto-duodenostomy, 469
Cholecysto-enterostomy in-Y, 130
Cholecysto-gastrostomy, 129
Cholecysto-jejunostomy, 469
Cholecystostomy, 448
in cirrhosis, 185
Cholecystotomy, 448
Choledochostomy, 128, 458
Choledochotomy, 119, 458
retroduodenal, 466
transduodenal, 120, 464
Choledocho-enterostomy, 127
statistics, 134
Choledochoplasty, 126
Choledochus, *see* Common duct.
drainage, 128
Cholesterin gall-bladder, 70
source, 10
Cholelithiasis, ætiology, age, 84
bacteria, 91
cardiac lesions, 90
climate, 89
exciting causes, 91
foreign body, 90
occupation, 89
parasites, 90
pregnancy, 88
race, 89
sepsis, 91
sex, 88
silk thread, 90
thyroid degeneration, 91

Cholelithiasis, cholecystectomy, 116
lesions, 68
pathology, 64
perforation of gall-bladder, 71
pericholecystitis, 71
predisposing causes, 84
simple, 69
symptomatology, 93
crepitation, 106
enlargement of gall-bladder, 104
fever, 105
symptoms, gastric, 94
hæmatemesis, 106
pain, 94
local, 94
referred, 99
tenderness, 101
vomiting, 106
treatment, 110
Carlsbad, 110
operation, contraindication, 114
Cirrhosis, liver, 182
ætiology, 183
biliary, 182
operations, 185
portal, 182
treatment, 185
Clay-colored stools, 33
Colic, biliary, treatment, 117
Collins's incision, 442
Common duct, atresia, 39
ball-valve obstruction, 33, 78
calculus, 75
treatment, 118
cyst of, 79
dilatation, 79
symptoms, 79
treatment, 80
drainage, 128
gall-stone in, 108
obstruction, 78, 79
congenital, 40
diagnosis, 109
symptoms, 108
resection, 126, 225
statistics, 133
stricture, 123
Concretions, bilary, *see* Gall-stones.
"Corset-liver," 157
Courvoisier's incision, 441
law, 80
violations, 81
Cyst, common duct, 79
liver, dermoid, 204
echinococcus, 189
hydatid, 189
ætiology, 189
diagnosis, 195
pathology, 190
symptoms, 193
treatment, 197
lymph, 204
Cyst, liver, non-parasitic, 203
parasitic, 189
retention, 204
pancreas, 368
ætiology, 368
diagnosis, 378
hydatid, 372
Körte's classification, 375
pathology, 369
proliferation, 371
retention, 369
symptoms, 373
traumatic, 371
treatment, 379
spleen, 400
blood, 401
diagnosis, 403
echinococcus, 400
lymph, 402
non-parasitic, 401
parasitic, 400
pathology, 402
retention, 402
serous, 401
simple, 401
symptoms, 402
treatment, 403
Cystic duct, calculus, treatment, 117
obstruction, diagnosis, 108
stricture, 123
Cystadenomata of liver, 204
Czerny's incision, 443

Depage's operation, 472
Dermoid cyst, liver, 204
Desjardins' point, 334
Diabetes, pancreatic, 270
Duct, common, atresia, 39
ball-valve obstruction, 33, 78
calculus, 75
cyst, 79
dilatation, 79
drainage, 128
obstruction, 78
congenital, 40
resection, 126, 225
stricture, 123
cystic, calculus, 117
obstruction, 108
stricture, 123
hepatic, calculi in, 83
stricture, 124
Ducts, biliary, *see* Bile-ducts.
Duodeno-choledochostomy, 466
Duodeno-choledochotomy, 466

Echinococcus, alveolaris, 191
cyst, liver, 189
spleen, 400
granulosus, 189
multilocularis, 191
scoleciparien, 191
Eck's fistula, 186

Empyema of gall-bladder, 52
Endothelioma of Gaucher, 408
Epiplopexy, in cirrhosis, 186
statistics, 187
Excision of choledochus, 225
papilla of Vater, 224

Fat necrosis in pancreatitis, 280
Fever in cholecystitis, 57
in cholelithiasis, 105
"steeple" chart, 106
Fistula, biliary, 144
external, 148
internal, 144
post-operative, 150
statistics, 144
Eck's, 186
mucous, 149
post-operative, 149
Floating liver, 159
Foreign bodies in gall-stones, 12

Gall-bladder, absence, congenital, 40
treatment, 41
bifid, 42
carcinoma, 217
pathology, 218
symptoms, 220
treatment, 220
cholesterin, 70
cystic degeneration, 217
disease, during typhoid, 21
double, 42
empyema, 52
enlargement in cholelithiasis, 104
"foreign body inflammation," 66
hemorrhage into, 56
hour-glass, 43
hydrops, 51, 75
injuries, 249
operations, 440
incisions, 441
perforation, 59, 71
during typhoid, 20
position, anomalies, 42
rupture, 249
treatment, 252
shape, anomalies, 42
stagnant, 50
strawberry, 117
typhoid infection, 19
volvulus, 55
wandering, 42
wounds, penetrating, 253
Gall-stones, bilirubin-calcium, 16
calcareous, 16
cholesterin, 15
color, 13
common, 16
formation of, 8
foreign bodies, 12
history, 8
pathogenesis, 9
Gall-stones, formation, rapidity, 12
hardness, 13
in common duct, 75, 108
treatment, 118
in cystic duct, symptoms, 107
treatment, 117
in hepatic duct, 83
laminated, 15
largest, 14
localization, 106
location, 67
mixed cholesterin, 16
mulberry, 14
number, 14
obstruction of intestines by, 152
obturation, 153
physical characters, 13
radial cholesterin, 15
recurrence of, 113
shape, 13
size, 14
varieties, 15
weight, 14
Gastro-splenic ligament, 384
Glycosuria, alimentary, 279
pancreatic, 270, 278
Gumma, liver, 208
Gunshot wounds, liver, 244

Hemorrhage in jaundice, 31
in pancreatitis, 282
into gall-bladder, 56
Hepatectomy, 475
Hepatic artery, aneurism, 140
ligation, 141
duct, calculi in, 83
stricture, 124
phlebotomy, 181
Hepatico-enterostomy, 127, 131
statistics, 135
Hepaticostomy, 128
Hepaticus drainage, 129
Hepatitis, suppurative, 164
Hepato-cholangeio-enterostomy, 128
statistics, 138
Hepatopexy, 163, 471
in cirrhosis, 186
Hepatoptosis, 159
diagnosis, 162
degrees, 161
pain, 162
partial, 158
symptoms, 161
treatment, 163
Hepatotomy, 120
for ascites, 185
in cirrhosis, 185
transpleural, 472
Hour-glass gall-bladder, 43
Hydatid cyst, liver, 189
ætiology, 189
complicated, treatment, 200
diagnosis, 195

Hydatid cyst, liver, marsupialization, 198
symptoms, 193
treatment, 197
pancreas, 372
fremitus, 194
Hydrops of gall-bladder, 51, 75
vesicæ felleæ, 51

Icterus, *see* Jaundice.
Incision, bayonet, 442
Bevan's, 445
Collins's, 442
Courvoisier's, 441
Czerny's, 443
Kausch's ,444
Kehr's, 442
Kocher's, 441
Körte's, 441
Langenbuch's, 442
Riedel's, 442
Robson's, 442
Sprengel's, 444
Tait's, 442
Wellenschnitt, 442
Infection of biliary tract, 3
Intestinal obstruction by gall-stones, 152

Jaundice, 28
adrenalin, 32
black, 357
blood changes, 31
coagulation time, 31
calcium chloride in, 32
catarrhal, 45
acute, diagnosis, 46
treatment, 44
cause, 28
classification, 34
congenital, 39
discoloration of skin, 29
extra-hepatic, 35
gelatin, 32
hemorrhage, 31
in carcinoma, bile-ducts, 222
pancreas, 356
in cholecystitis, 58
in pancreatic calculus, 347
disease, 280
in pancreatitis, chronic, 329
infantile, 39
intermittent, 78
itching of skin, 30
non-obstructive, 34
obstructive, 35
serum therapy, 32
significance, 34
stools, 33
thyroid extract, 32
urinary changes, 31

Kausch's incision, 444
Kehr's incision, 442
Kehr's operation, 129
Kocher's incision, 441
Körte's incision, 441
Kidney, movable, in biliary obstruction, 139

Langenbuch's incision, 442
Leptothrix, 3
Lieno-phrenic ligament, 385
Lieno-renal ligament, 385
Ligament, gastro-splenic, 384
lieno-phrenic, 385
lieno-renal, 385
Ligaments, spleen, 384
Ligation, hepatic artery for aneurism, 141
portal vein in cirrhosis, 186
Lipomatosis, 326
Liver, adenoma, 206
symptoms, 207
treatment, 208
angeiomata, 206
abscess, 164
ætiology, 165
amœbic, 168
diagnosis, 178
pain, 175
appendicular, 166
classification, 165
diagnosis, 175
exciting causes, 169
hepatotomy, 472
pathology, 169
post-operative treatment, 474
prognosis, 179
pyæmic, 165
symptoms, 172
traumatic, 165
treatment, 180
tropical, 168
carcinoma, 211
diagnosis, 214
pathology, 212
symptoms, 213
treatment, 215
cirrhosis, 182
ætiology, 183
biliary, 182
operations for, 185
portal, 182
treatment, 185
corset, 157
cyst, 189
dermoid, 204
echinococcus, 189
hydatid, 189
diagnosis, 195
symptoms, 193
treatment, 197
lymph, 204
non-parasitic, 203
parasitic, 189
retention, 204

Liver, cystadenomata, 204
enlargement, 82
fibromata, 206
floating, 159
gumma, 208
injuries, 235
operations, 236
linguiform lobulation, 158
movable, 159
acquired, 160
congenital, 159
myomata, 206
operations, 471
position, anomalies, 157
Riedel's lobe, 158
rupture, 237
diagnosis, 240
omentoplasty, 244
symptoms, 238
treatment, 241
sarcoma, 210
shape, anomalies, 157
size, anomalies, 157
surgery, 157
tuberculoma, 210
tumors, 206
malignant, 210
wounds, gunshot, 244
treatment, 246
stab, 247
treatment, 247
"Luschka's Gänge," 54
Lymphadenoma, splenic, 408
Lymph-nodes, pancreatic, inferior, 315
superior, 315
pancreatico-duodenal, 315
pancreatico-splenic, 315

Marsupialization of hydatid cyst, 198
Morison operation, 186
Movable kidney in biliary obstruction, 139
liver, 159
spleen, 396
diagnosis, 398
pathology, 397
symptoms, 397
treatment, 399
Mucous fistula, 149
Mulberry stones, 14
Müller's test in pancreatitis, 276
Mumps in pancreatitis, 260
Muscular rigidity in cholecystitis, 57

Obliteration of bile-ducts, congenital, 39
Obturation, gall-stone, 153
Obstruction, biliary passages, 122
by aneurism, 140
external, 139
common duct, congenital, treatment, 40
cystic duct, 108
Obstruction, intestinal, gall-stone, 152
diagnosis, 154
treatment, 155
Olive oil breakfast, 63
Omentopexy in cirrhosis, 186
Omentoplasty in rupture liver, 244
Operation, cholecystectomy, 454
cholecystendysis, 447
cholecystenterostomy, 129, 469
cholecysto-colostomy, 469
cholecysto-duodenostomy, 469
cholecysto-gastrostomy, 129
cholecysto-jejunostomy, 469
cholecystostomy, 448
cholecystotomy, 447
choledocho-enterostomy, 127
choledochoplasty, 126
choledochostomy, 128, 458
choledochotomy, 458
retroduodenal, 466
transduodenal, 464
Depage's, 472
drainage, common duct, 128
hepatectomy, 475
hepatico-enterostomy, 127
hepaticostomy, 128
hepaticus drainage, 129
hepatopexy, 163, 471
hepatostomy, 129
hepatotomy, transpleural, 472
Kehr's, 129
Morison's, 186
omentopexy, 186
omentoplasty, 244
pancreatectomy, 477
pancreato-enterostomy, 480
pancreatostomy, 349
resection choledochus, 126
splenectomy, 481
splenopexy, 482
splenotomy, 482
Talma's, 186
Terrier's, 456
Operations, technique, 440
preparation of patient, 440

Pain in amœbic abscess, 175
in carcimona of bile-ducts, 222
of liver, 214
of pancreas, 357
of stomach, 101
in cholecystitis, 56
in cholelithiasis, 94
in cyst of spleen, 402
in gumma of liver, 209
in hepatoptosis, 162
in pancreatic calculus, 346
cyst, 373
disease, 285
pancreatitis, acute, 294
chronic, 328
in ruptured spleen, 424
in splenitis, 405

Pain in ulcer of duodenum, 100
of stomach, 100
Pancreas, abscess, 307
treatment, 308
accessory, 256
adenoma, 367
annulare, 257
anomalies, congenital, 256
atrophy, 259
carcinoma, 353
diagnosis, 362
operation, 365
pathology, 354
symptoms, 356
treatment, 365
cyst, 368
ætiology, 368
diagnosis, 378
hydatid, 372
Körte's classification, 375
pathology, 369
proliferation, 371
retention, 369
symptoms, 373
traumatic, 371
treatment, 379
disease of, 259
azotorrhœa, 276
diagnosis, 283
fat necrosis, 280
glycosuria, 278
hemorrhage, 282
jaundice, 280
Müller's test, 276
physical signs, 280
predisposing causes, 284
Sahli's test, 276
Salomon's test, 277
symptoms, 274, 285
displacement, 258
divisum, 257
gangrene, 307
infection, 4, 259
injuries, 351
minus, 257
operation, 477
ring-formed, 257
rupture, 351
sarcoma, 367
surgery, history, 254
triangle of infection, 264
tumors, 353
wound, gunshot, 351
stab, 351
Pancreatectomy, 475
Pancreatic apoplexy, 255, 299
calculus, 268, 346
diagnosis, 348
operation, 348
symptoms, 346
treatment, 348
diabetes, 270
glycosuria, 270
Pancreatic infantilism, 259
lymphangeitis, 266, 314
lymph-nodes, inferior, 315
superior, 315
point, 334
reaction, 336
of Cammidge, 278
steatorrhœa, 274
Pancreatico-duodenal lymph-nodes, 315
Pancreatico-hepatic area, 334
Pancreatico-splenic lymph-nodes, 315
Pancreatitis, acute, 288
ætiology, 291
diagnosis, 297
pathology, 292
prognosis, 299
statistics, 303
symptoms, 294
treatment, 303
chronic, 313, 323
ætiology, 323
catarrhal, 313
classification, 339
diagnosis, 326, 338
interacinar, 325
interlobular, 324
pathology, 324
prognosis, 340
symptoms, 326
treatment, 341
urine, 336
hemorrhagic, 289
subacute, 307
diagnosis, 307
treatment, 308
operation, 308
suppurative, 307
Pancreato-enterostomy, 480
Pancreatostomy, 349
Papilla of Vater, excision, 224
Paracentesis for ascites, 185
Paratyphoid bacillus, 17
Perforation of gall-bladder, 59
Pericholecystic abscess, 71
adhesions, 53
Pericholecystitis, 53, 71
suppurative, 53
Peritonitis, biliary, 53
Pneumokystes hydatiques, 193
Portal thrombosis, 142
Preparation for operation, 440
Pseudohæmophilia, 31
Pseudoleukæmia, splenic, 408

Ranula pancreatica, 370
Resection, choledochus, 126
Riedel's incision, 442
lobe, 158
Robson's incision, 442
point, 101, 334
Rupture, liver, 237
omentoplasty, 244
pancreas, 351

Rupture, spleen, 421

Sahli's test in pancreatitis, 276
Salomon's test in pancreatitis, 277
Sarcoma, liver, 210
  pancreas, 367
  spleen, 403
Serum sickness, 32
  therapy in jaundice, 32
Sialodochitis pancreatica, 313
Sialorrhœa pancreatica, 278
"Simple cholelithiasis," 69
Spleen, abscess, 404
    diagnosis, 406
    pathology, 405
    symptoms, 405
    treatment, 407
  absence, 385
  accessory, 385
  anatomy, 383
  anomalies, 385
  blood-vessels, 384
  chondroma, 403
  cyst, 400
    blood, 401
    diagnosis, 403
    echinococcus, 400
    lymph, 402
    non-parasitic, 401
    parasitic, 400
    pathology, 402
    retention, 402
    serous, 401
    simple, 401
    symptoms, 402
    treatment, 403
  dislocated, 396
  ectopic, 396
  enlargements, 390
    ætiology, 395
    diagnosis, 391
  fibroma, 403
  floating, 396
  hypertrophy, splenectomy, 427
  injuries, 421
    operation, 425
  ligaments, 384
  lymphangeioma, 403
  movable, 396
    ætiology, 396
    diagnosis, 398
    pathology, 397
    symptoms, 397
    treatment, 399
  movements, 389
  operations, 427, 481
  osteoma, 403
  physiology, 386
  prolapse, 396
  relations, 385
  rupture, 421
    diagnosis, 424
    pathology, 422
Spleen, rupture, symptoms, 423
    treatment, 425
  sarcoma, 403
  surgery, 383
  tumors, 403
  tuberculosis, 404
  wandering, 396
  wounds, 423
Splenectomy, 427, 481
  blood-changes, 386
  in splenic anæmia, 430
  prognosis after, 434
Splenic anæmia, 408
  lymphadenoma, 408
  pseudoleukæmia, 408
Splenitis, suppurative, 404
Splenocleisis, 420
Splenomegaly, cryptogenic, 408
  hæmolytic, 418
  primary, 408
Splenopexy, 482
  in cirrhosis, 186
Splenoptosis, 396
Splenotomy, 482
Sprengel's incision, 444
Stab wounds, liver, 247
Stagnant gall-bladder, 50
Staphylococcus pyogenes albus, 3
    aureus, 3
Steatorrhœa in pancreatitis, 274
"Stone-forming catarrh," 8
Strawberry gall-bladder, 117
Streptococcus pyogenes, 3
Stricture of bile-ducts, congenital, 39
    treatment, 124
  common duct, 123
  cystic duct, 123
  hepatic duct, 124

Tænia echinococcus, 189
Tait's incision, 442
Talma operation, 186
Talma-Drummond operation, 186
Tenderness in Boas's area, 103
  cholecystitis, 57
  cholelithiasis, 101
Terrier's operation, 456
Thrombosis, portal, 142
Tuberculoma, liver, 210
Tuberculosis, spleen, 404
Typhoid bacillus, 17
  carriers, 7, 26
    treatment, 26
  fever, gall-bladder complications, 20
    disease, 21
  infection of biliary tract, 17

Urine, bile in, 30

Visceropexy in cirrhosis, 186
Volvulus of gall-bladder, 55

Vomiting in cancer of stomach, 106
cholecystitis, 57
cholelithiasis, 106
ulcer, duodenal, 106
gastric, 106

Wandering gall-bladder, 42
Wounds, gunshot, liver, 246
stab, liver, 247

Xanthelasma, 30
Xanthoma, 30